









# **THE DICTIONARY OF PRACTICAL MEDICINE**



# THE DICTIONARY OF PRACTICAL MEDICINE

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IN THREE VOLUMES

*WITH 48 PLATES AND 109 FIGURES IN THE TEXT*

**VOL. III**

**PREGNANCY, PERNICIOUS VOMITING OF, to ZONA**

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# LIST OF PLATES

## VOL. III

PLATE	FACING PAGE
6. GUTTATE PSORIASIS WITH TYPICAL LESIONS OVER ELBOW . . . . .	16
27. PSORIASIS WITH CIRCINATE SCALY PATCHES . . . . .	17
28. FUNGOUS PARASITES . . . . .	188
29. TINEA TONSURANS AND SUPPURATING TINEA CIRCINATA OF ARM . . . . .	140
30. RODENT ULCER . . . . .	203
31. LUPUS VULGARIS . . . . .	208
32. SMALLPOX . . . . .	216
33. TO SHOW DISTRIBUTION OF THE DORSAL SPINAL ROOTS TO THE SKIN . . . . .	286
34. HARD CHANCRE . . . . .	301
35. MACULO-PAPULAR SYPHILIDE ( <i>Colour</i> ) . . . . .	304
36. PAPULAR SYPHILIDE . . . . .	306
37. PUSTULAR SYPHILIDE . . . . .	308
38. PARASITE-BEARING TSETSE FLIES AND TICKS ( <i>Colour</i> ) . . . . .	366
39. UNORGANIZED DEPOSITS IN URINE . . . . .	454
40. ORGANIZED DEPOSITS IN URINE . . . . .	456
41. DIAGRAM OF THE VEGETATIVE NERVOUS SYSTEM ( <i>Colour</i> ) . . . . .	510
42. RADIOGRAMS . . . . .	547
43. „ . . . .	550
44. „ . . . .	551
45. „ . . . .	556
46. „ . . . .	557
47. „ . . . .	562
48. „ . . . .	563



# LIST OF FIGURES IN THE TEXT

## VOL. III

FIG.	PAGE
84. Lillingston's apparatus for artificial pneumothorax . . . . .	57
85. <i>Spironema recurrentis</i> in blood-film . . . . .	100
86. Brünings' laryngoscope, with Paterson's forceps in position . . . . .	106
87. <i>Schistosoma hæmatobium</i> , male and female . . . . .	159
88. Fork-tailed cercariæ . . . . .	159
89. The sigmoidoscope . . . . .	197
90. Microcephaly . . . . .	210
91. To show sensory conduction in spinal cord . . . . .	235
92. Maddox rod . . . . .	283
93. Method of suturing flat tendons . . . . .	334
94. „ „ cylindrical tendons . . . . .	334
95. Incision for ingrown toe-nail . . . . .	346
96. <i>Trichinella spiralis</i> . . . . .	359
97. <i>Trypanosoma gambiense</i> . . . . .	367
98. „ „ <i>rhodesiense</i> . . . . .	367
99. Schema of <i>trypanosoma</i> . . . . .	367
100. Anterior urethroscope . . . . .	432
101. Posterior „ . . . . .	433
102. Collapsing pulse of aortic incompetence due to acute endocarditis . . . . .	492
103. Pulse of aortic incompetence in later life due to degenerative change . . . . .	492
104. Pulse of aortic stenosis . . . . .	495
105. „ „ extreme mitral stenosis . . . . .	501
106. Operation for subcutaneous whitlow . . . . .	531
107. Incisions for removal of nail . . . . .	532
108. „ „ exposing tendons of hand, etc. . . . .	533
109. Tibial periosteal nodes, ulcers, and deformed phalanges . . . . .	565





# THE DICTIONARY OF PRACTICAL MEDICINE

## VOL. III

**PREGNANCY, PERNICIOUS VOMIT-  
ING OF** (*see* VOMITING, PERNICIOUS,  
OF PREGNANCY).

**PREGNANCY, POST-MORTEM AP-  
PEARANCES OF** (*see* ABORTION, CRIM-  
INAL).

**PREGNANCY, SIGNS AND SYMPTOMS  
OF.**—The diagnosis of pregnancy is arrived  
at by the recognition of certain symptoms and  
physical signs which vary according to its  
duration. In most cases diagnosis presents  
no difficulties, especially in the later months,  
for the signs are obvious and unmistakable.  
In a few, however, they may be obscure and  
indeterminate, so that a positive diagnosis is  
not possible.

Recently, a method of serum diagnosis has  
been described which may prove to be useful as  
a supplementary aid to clinical examination,  
but as yet there is not sufficient evidence of  
its absolute value, whilst its great practical  
difficulties of technique render it impossible to  
apply as a routine measure. (*See* Abderhalden  
Reaction, under SEROLOGICAL DIAGNOSIS.)

The **signs and symptoms** of pregnancy may  
be classified into two groups—(1) presumptive,  
(2) absolute. Although the recognition of the  
former in any given case may evoke a strong  
suspicion, bordering on certainty, of the  
correctness of a positive diagnosis, it is never  
possible to give this opinion based upon the  
presumptive signs above.

With further clinical knowledge of gynæco-  
logical conditions, it is clear that the few so-  
called "absolute" signs of pregnancy are  
diminishing to such an extent that foetal  
movements and the foetal heart-beat may be  
said to constitute the only two certain signs.

**The first three months.**—The first indication  
of pregnancy is *amenorrhœa*, characteristically  
sudden and complete, occurring in a woman  
of sound health. Pathological suppression of  
the menses is usually associated with other  
symptoms which suggest the cause, but there  
are some cases of pathological amenorrhœa  
of sudden onset which exactly simulate the  
physiological amenorrhœa of pregnancy. Such  
cases often originate in mental disturbance,  
anxieties, and preoccupations, and time alone  
can settle the diagnosis. The fact remains that  
a sudden menstrual suppression occurring in a  
healthy married woman between 20 and 40 is  
strong presumptive evidence of pregnancy.

At the time of the first missed period the  
woman frequently notices that the mammary  
sensations and the swelling of the breasts are  
more distinct. Mammary discomfort, notice-  
able for the first time at this early period, often  
remains for the first two or three months,  
especially in primigravidæ.

During the second month *morning vomiting*  
makes its appearance, usually about a fortnight  
after the first missed period. Its incidence  
is by no means constant, and it is variable  
in degree. Typically, some nausea is felt on  
waking in the morning, followed by retching  
once or twice on rising, whilst during the  
remainder of the day there is no return of the  
symptom.

Many pregnant women, especially multiparæ,  
never complain of sickness, and not more than  
80 per cent. of primigravidæ suffer, whereas  
others may be subject to severe vomiting  
bordering on the pathological. Morning sick-  
ness usually persists for about six or eight  
weeks, but the duration may be as short as  
three weeks or as long as five or six months.

## PREGNANCY, SIGNS AND SYMPTOMS OF

There are no associated gastric symptoms: the tongue is clean, the appetite is not greatly disturbed after breakfast, and there is seldom any accompanying dyspepsia. Morning vomiting due to other causes is not often met with in young women below 30, as it is usually due to chronic dyspepsia and gastritis, or alcoholism. These conditions produce other signs pointing to their origin.

A further and minor symptom, sometimes complained of during the second month, is *frequency of and slight pain on micturition*. It is due to the bulky uterus becoming more anteverted and pressing down upon the bladder, causing irritability. It is generally transient, of trivial importance, and associated with no evidence of cystitis.

The *objective* signs during the first three months are few, but important. The first to appear, and the most characteristic, is the *softening of the vaginal cervix*. It is a gradual process, first detectable about the sixth week, and commencing in the zone immediately bordering on the external os. Afterwards it rapidly extends through the whole thickness of the vaginal cervix, and is complete during the third month. Occasionally, softening is incomplete, and scarcely noticeable, and, on the other hand, some non-pregnant conditions, such as vascular fibro-myoma and subinvolution, are associated with it.

*Enlargement of the uterus* is difficult to detect before the eighth, and impossible to detect before the sixth week. It is discovered by careful bimanual examination, which reveals the body enlarged in a manner almost peculiar to pregnancy. The fundus uteri appears to be the portion chiefly concerned in the enlargement, whereas the lower part of the body and the cervix are of normal size. This condition is distinct from enlargement due to endometritis, when the whole of the body of the uterus is uniformly increased in size.

The recognition of the above feature leads the obstetrician to appreciate *Hegar's sign*—a valuable accessory aid to diagnosis which is available from the sixth to the twelfth weeks. During this period the fundus only is occupied by the ovum, while the lower uterine segment is empty, and, moreover, softened. Hence, if the fingers of the two hands in a bimanual examination are made to approximate each other through the soft lower segment, the fundus can be felt, enlarged and firm, in sharp contrast with the soft unoccupied lower segment, through which the fingers will almost meet. There is no

other condition which typically yields this sign, but in rare cases a small single fundal fibroid may simulate the earliest enlargement due to pregnancy.

Distension of the vulval veins gives rise to *violet discoloration of vulva*.

*Mammary signs* develop steadily during the first three months. At an early period there is an hypertrophy of the peripheral lobules, which become distinctly palpable, with a corresponding enlargement of the whole organ. Superficial veins become prominent, and in the third month it is possible to express a turbid serum from the nipple.

Thus, by the end of the third month the pregnant woman has missed three menstrual periods, while the breasts have become enlarged and tender, and there may have been some morning sickness. Physical examination shows that the uterus is enlarged to about the size of an orange, rounded and intrapelvic, with the cervix much softened.

**The second three months.**—The middle period of pregnancy affords additional fresh symptoms and signs, some of which are of great significance. The most prominent is "*quickening*," first noticed in the seventeenth week. Its time of onset is peculiarly constant. True quickening is a sure symptom of pregnancy, but it can be simulated by and mistaken for intestinal movements, and, being subjective, cannot be relied upon as a positive indication. On the other hand, in rare cases it may not be felt by the pregnant woman.

*Pigmentation of the skin* begins to appear in the fourth month, being especially pronounced in the areola of the nipple, along the linea nigra—i.e. the median line between the navel and the pubes—in the axillæ, and under the eyes. The *secondary areola*, a pigmented zone surrounding the primary areola, makes its appearance much later, towards the end of pregnancy. It is a faint, splashed brownish stain, especially seen in brunettes, and is highly characteristic.

During this part of the gestation period four new and important physical signs can be recognized. From the sixteenth week onwards *abdominal enlargement* is evident, and from the sixteenth to the twenty-eighth *internal ballotement*, due to a body floating within the uterus, can be elicited by internal examination. The finger is placed in the posterior fornix while the woman lies upon her back. A smart upward impulse transmitted to the uterine body is followed after an interval of a second by the sensation of a light tap, produced by the impact

## PREGNANCY, SIGNS AND SYMPTOMS OF

of the foetus as it falls back again on to the finger. It is an extremely important sign, and is almost, if not quite, a certain indication of pregnancy. It is possible that it may be imperfectly imitated by a degenerated fibroid containing small masses floating in a cystic cavity. Such fibroids must be excessively rare.

The third fresh sign is that of *intermittent contraction of the abdominal swelling* formed by the pregnant womb. It can be detected towards the end of this period, as soon as the uterus causes a definite abdominal enlargement. It has been said that an abdominal tumour which can be felt to harden and soften intermittently must be the gravid uterus, but, although this is true in the great majority of cases, the sign is not absolutely pathognomonic, for a uterus containing a large cystic fibroid can produce intermittent contractions very closely similar. When an irregular mass exists in the abdomen, the recognition of intermittent contractions of a part of the tumour is important, for it is strong evidence that that part is an enlarged and gravid uterus, while the remainder may be fibro-myoma.

The fourth and most important sign is the *fœtal heart-beat*, which can often be heard as early as the twentieth week. At this early stage the first sound only may be detected. From the twentieth to the twenty-eighth weeks the fœtal heart is most easily heard at a point midway between the umbilicus and the pubes, whereas after this period the site of maximum intensity depends upon the lie of the child. Thus, in most vertex and breech presentations it is heard without difficulty, in the former just above the outer half of Poupart's ligament, in the latter at the level of the umbilicus. In shoulder presentations the heart is heard with difficulty if the child is in the dorso-posterior position; and when the presentation is by the face the heart may be heard loudest on the side opposite to the back, i.e. on the same side as the small parts, owing to the projection forwards of the fœtal chest against the uterine wall.

**The last three months.**—Diagnosis of pregnancy during the seventh, eighth, and ninth months is usually very simple. A fresh train of symptoms appears, referable to the presence of a large mass in the abdomen causing pressure on veins, nerves, diaphragm, and viscera. Thus, the woman may complain of varicose veins, or piles, while cramps and neuralgia in the lower limbs are extremely common. Constipation, dyspepsia, and frequency of micturition are prone to occur, though there is often consider-

able amelioration of these symptoms during the last two weeks, when the uterus sinks into the pelvis. The abdomen is enlarged by an *ovoid tumour* rising out of the pelvis, the chief characteristics of which are as follows: (a) Its *outline* is regular, only slight and transient prominences due to the projection of fœtal limbs being present. (b) There is *intermittent hardening and softening*, the wave of contraction lasting for about a minute, after which the uterus gradually relaxes, and remains soft for two or three minutes. (c) It is of *elastic consistence*, i.e. it is not fluctuant, neither is there a thrill. Exceptions to the normal occur in hydramnion, where there is often a pronounced thrill, which is equally distributed over the whole tumour, in contradistinction to multi-locular ovarian tumours, where the thrill is uneven in quality and is differently distributed in different regions. (d) *Fœtal parts* can, as a rule, be recognized by careful palpation. They are easily discovered in thin women, in multiparæ, and when there is the normal or less than normal amount of liquor amnii. On the other hand, they are sometimes difficult to detect in fat women and primigravidæ, in hydramnios, in pregnancy complicated by fibroids which obscure the anterior uterine wall, and in cases in which the uterus is in an irritable and tense condition. As has been pointed out, a uterus which is enlarged by a degenerated and cystic fibroid may almost exactly convey the impression that it contains fœtal parts, especially when it is felt to harden and soften under the hand. (e) *Fœtal movements* are frequently felt and seen, and heard on auscultation. When they are recognized, the evidence of pregnancy with a living child is absolute.

**Auscultation of the uterus** in late pregnancy reveals four different sounds: (1) The fœtal heart-beat. (2) The uterine souffle—a blowing bruit, synchronous with the internal heart-beat, especially well heard above the inner half of Poupart's ligament. It is produced by the blood-flow through the enlarged and tortuous uterine arteries. This sound is not peculiar to pregnancy, for it is commonly heard in cases of large and vascular fibroids. It can be heard as early as the fourth month, when it is a most useful sign. (3) Fœtal movements are audible as dull thuds, and are often heard some weeks before they can be seen or felt. This is a valuable sign, as it yields evidence of a positive nature at an early stage. (4) The funic souffle is the bruit of the umbilical artery traversing the cord, and is only heard when the bell of the

## PREGNANCY, SIGNS OF

stethoscope happens to compress the cord against the child's back. It is very rarely detected.

**Vaginal examination** at this period is directed towards determining (1) the condition of the cervix, whether soft or hard, closed or patulous; (2) whether the abdominal tumour and the cervix are one and the same mass, i.e. confirming the diagnosis that the abdominal mass is a uterine swelling; (3) the contents of the lower uterine segment, whether foetal head, breech, or other part.

By ordinary careful clinical examination, then, it is nearly always possible to make a positive diagnosis of pregnancy at least after the twenty-fourth week, and in many cases as early as the sixteenth week.

The conditions most liable to be mistaken for pregnancy in the *first three months* are—(1) Subinvolution, associated with the amenorrhœa of lactation. Here we find the uterus enlarged and the cervix soft, together with amenorrhœa. The enlargement, however, is not often greater than that of ten weeks' pregnancy, and the shape is not characteristic. In many cases the diagnosis must be postponed until additional signs develop. (2) A single fibroid uniformly enlarging the uterus. (3) Endometritis and chronic metritis.

In the *later months*, when the uterine enlargement forms an abdominal swelling, the condition may have to be distinguished from fibroids, ovarian cysts, and even from an over-distended bladder. The recognition of pregnancy and coexisting fibroids or ovarian tumours may be extremely difficult. It is especially in such cases that the Abderhalden serum test is of value.

A. W. BOURNE.

**PREMATURE CONTRACTIONS** (*see* HEART-BEAT, ABNORMALITIES OF).

**PREPUTIAL CALCULI** (*see* URINARY CALCULI).

**PRESBYOPHRENIA** (*see* DEMENTIA, SENILE).

**PRESBYOPIA** (*see* REFRACTION AND ACCOMMODATION, ERRORS OF).

**PRIAPISM** (*see* SEXUAL FUNCTIONS, MALE, DISTURBANCES OF).

**PRIMARY LATERAL SCLEROSIS** (*see* LATERAL SCLEROSIS, PRIMARY).

**PROIDENTIA** (*see* PELVIC ORGANS, FEMALE, DISPLACEMENTS OF).

## PROGERIA

**PROCTITIS**.—Inflammation of the rectum results from the presence of foreign bodies or from infection by micro-organisms. It is not always possible to isolate a specific organism, but the bacilli or amœbæ of dysentery, the gonococcus, the spirochæta of syphilis, and the ova of schistosoma are occasional specific causes. Septic micrococci may also be responsible.

The **symptoms** are pain on defæcation, tenesmus, a sensation of fullness and discomfort in the perineal region, a feeling after the bowels have been opened that something still remains to be evacuated, and a discharge of pus, mucus, or bloodstained material from the rectum. Constipation is the rule.

**Diagnosis** is usually clear on local examination. Piles, fissure and fistula must be excluded. Digital and proctoscopic examination will show either a swollen inflamed mucous membrane or possibly a congested and perhaps ulcerated area of the rectum. Microscopical or cultural investigation of the discharge may throw light upon the exciting cause.

**Treatment**.—Any specific condition must be specially treated, whether it be dysentery, schistosomiasis or syphilis. In the absence of any special cause it is necessary to keep the bowels loose by suitable aperients, and to relieve the pain and discomfort either by fomentations applied to the perineum, or by hot sitz-baths, or by gently washing out the rectum with warm saline solution or dilute acetate-of-lead lotion. A suppository containing  $\frac{1}{4}$  gr. of morphine tartrate may be inserted at night if the pain is very severe. Careful examination must be made to see that no stricture develops as the condition subsides.

ZACHARY COPE.

**PROGERIA** (Premature Senility).—Progeria is the name given by Hastings Gilford to a very rare affection described by him in which senile changes occur during early life. In the most extreme examples, such as those described below, a most striking condition is seen, but the interest of progeria is by no means confined to such instances as these, which are excessively rare. Deeper researches by Gilford and others, not only upon individuals, but upon separate organs and tissues, seem to suggest the possibility of a general law that tissues in which development has been retarded (infantilism) tend to undergo premature senile degeneration.

The two extreme cases which are referred to, both arose during the first few years of life.

## PROSTATE, NEW GROWTHS OF

The boys were remarkably alike; stunted in growth but having the appearance of extreme old age by the time they were adolescent. Both died before reaching the age of twenty-one, one from syncope, the other from angina. Their faces were old and shrivelled, their bodies devoid of fat, their arteries atheromatous and calcified, their hair sparse and white. Their voices were high-pitched and the gait and posture in each case were those of the extremely old. Post mortem, in addition to the arterial changes, the kidneys were fibrosed and the adrenals and intestines atrophic.

Of less extreme cases may be mentioned one of premature greyness of the hair occurring in a case of infantilism (Caldwell).

REGINALD MILLER.

**PROGRESSIVE LENTICULAR DEGENERATION** (*see* LENTICULAR DEGENERATION, PROGRESSIVE).

**PROGRESSIVE MUSCULAR ATROPHY** (*see* MUSCULAR ATROPHY, PROGRESSIVE).

**PROGRESSIVE MUSCULAR DYSTROPHY** (*see* MYOPATHY).

**PROGRESSIVE NEURO-MUSCULAR ATROPHY** (*see* MUSCULAR ATROPHY, PERONEAL).

**PROLAPSE OF RECTUM** (*see* RECTUM, PROLAPSE OF).

**PROLAPSE OF URETHRA** (*see* URETHRA, PROLAPSE OF).

**PROLAPSE OF UTERUS** (*see* PELVIC ORGANS, FEMALE, DISPLACEMENTS OF).

**PROLAPSE OF VAGINA** (*see* PELVIC ORGANS, FEMALE, DISPLACEMENTS OF).

**PROPTOSIS** (*see* ORBIT, AFFECTIONS OF).

**PROSTATE, NEW GROWTHS OF.**—The new growths considered under this title are (A) Adenoma and (B) Carcinoma.

A. ADENOMA (SO-CALLED HYPERTROPHY)

**Pathology.**—Of those enlargements of the prostate which are due to new growth, 90 per cent. are innocent in character, and are adenomata, which may be encapsuled or non-encapsuled.

**The encapsuled variety.**—The greater proportion of the adenomata possess definite capsules which they have formed for themselves

out of the natural stroma of the prostate. As seen in the early stages in a transverse section across the gland, an adenoma is a small yellowish body lying in the medulla. The common site of origin is in the interval between the converging ejaculatory ducts behind the urethra, and the capsule can be seen with the naked eye as a number of circular white strands surrounding a yellowish central body. Soon, other small adenomata appear in the neighbourhood of the primary one, and ultimately fuse together to form one mass containing in its centre the urethra, and surrounded by a very definite laminated capsule composed of all the tissues of the condensed cortex of the prostate. The line of cleavage in prostatectomy lies in this capsule, and the tumour removed consists, not of the whole prostate, but of one or more grouped adenomata containing the urethra.

As the prostate enlarges it projects backwards into the rectum, or upwards into the bladder, or in both directions. Behind the projection upwards into the bladder a post-prostatic pouch gradually forms, and it is in the stagnant urine contained in this that alkaline decomposition occurs and secondary phosphatic stones are formed. Small dilated veins appear beneath the attenuated mucous membrane surrounding the internal meatus, and often give rise to hæmaturia.

Microscopically, these tumours are composed of glandular acini lying in a very abundant stroma of fibrous tissue and unstripped muscular fibres.

**The non-encapsuled variety, or "fibrous" prostate.**—This is a more slowly growing, harder form of tumour, and the prostate very rarely shows any great increase in size. The enlargement takes the form of a slightly raised collar around the internal meatus. There is no definite laminated capsule as in the former type, and the adenomatous change is a more general one throughout the whole substance of the gland.

**Symptoms.**—Adenomata of the prostate rarely cause symptoms in men under 50, and the cases usually come under treatment for one of the following conditions:

(a) *Vesical irritability.*—In this variety the patient is apparently in the best of health, but for some months or years has had to rise once or twice in the night or early morning to pass water. There is also increased frequency of micturition in the daytime and perhaps slight urgency, but this may often pass unnoticed. A certain amount of hesitation in beginning to

## PROSTATE, NEW GROWTHS OF

empty the bladder, which is ultimately effected by a slightly less forcible stream than formerly, is usually present. The urine is clear, and the total amount passed in twenty-four hours may be increased.

(b) *Acute retention*.—Acute retention may occur at any time in a patient who for some years has experienced symptoms as in (a), but this accident may happen to a man who previously has not noticed any change in micturition. With the distension of the hypertrophied bladder there is much colicky suprapubic pain and straining.

(c) *Chronic retention*.—Here, the patient is a broken-down, shaky old man, who complains that he cannot hold his water. The tongue is coated, and he usually suffers from great thirst and other symptoms of renal inadequacy. Foul urine continually dribbles away from him, or is passed in small quantities at intervals of a very few minutes. In reality this is a case of painless distension and overflow, with the bladder often reaching as high as the umbilicus.

**Symptoms in detail.** (1) *Increased frequency of micturition*.—This is one of the earliest symptoms in prostatic disease, and is equally great night and day, although, naturally, it is more noticeable during the former.

(2) *Character of micturition*.—The chief characteristics are the difficulty the patient experiences in beginning micturition and the fact that straining hinders rather than helps the process. Although when the desire arises the call may be urgent, yet a delay of several minutes may elapse before the feeling can be gratified. The stream varies between one of diminished calibre only, and a mere dribble, and may intermit. The projection is poor, and micturition may end in a dribble which continues for several minutes.

(3) *Hæmaturia*.—Bleeding of the vesical type is not an infrequent symptom, and may come on spontaneously or follow the passage of a catheter. It is most frequently seen in innocent cases. It rarely attains serious proportions, and varies between a slight general ooze and a fairly profuse hæmorrhage from a small ruptured vein in the bladder neck.

(4) *Pain*.—Apart from acute retention of urine, pain is not a prominent feature, and usually amounts to little more than a feeling of fullness and weight in the perineum. Cases complicated by cystitis or stone show the characteristic pain of these conditions, and if pronounced renal stasis be present there will be an almost constant ache in the lumbar region.

(5) *Disorders of the sexual function*.—Stated briefly, the sexual changes are increased desire and a diminished power of gratifying it.

**Diagnosis**.—The above symptoms may be closely mimicked in some cases of organic nervous disease, such as *tabes dorsalis*, and a correct diagnosis of prostatic obstruction must be based upon physical signs. It is wise to begin by looking for evidence of a distended bladder and for tenderness in the renal regions. Per rectum, the prostate may be felt bulging into the anterior wall of the bowel, a slight exaggeration of the natural median groove giving the organ a bilobed character. Its natural contour is retained, and one of the most important points in the diagnosis of innocent enlargement is the preservation of its borders, especially the upper concave margin. Above the latter, unless the prostate is an unusually large one, the soft bladder base ought to be appreciated. The posterior surface is smooth and, in the encapsuled variety, the gland feels softer and more elastic, and is at the same time more movable than normal. Bimanual examination is of the greatest importance in estimating the real size of the prostate. It is essential also to remember that an adenoma may project upwards into the bladder and cause marked symptoms of obstruction while there is little or no evidence of enlargement to be detected from the rectal aspect.

The patient having micturated, a large coude catheter should be passed with the most rigid aseptic precautions, and the amount and character of the residual urine noted. The distance it is necessary to pass the catheter in before water is drawn off is some help in estimating the amount of intravesical projection. A short-beaked sound will give some idea of the character of the enlargement, but the cystoscope is more useful for this purpose.

**Complications**.—These are chiefly the results on the bladder and kidneys of the obstruction to the urinary outflow. The bladder becomes greatly hypertrophied, and, in consequence of the ensuing obstruction at the vesical orifice of the ureter, some dilatation of the pelvis with absorption and sclerosis of the renal parenchyma occurs. The organisms from an infected bladder readily pass upwards to the kidneys, and the pyelitis or pyelonephritis which follow may at any time progress and produce a fatal result from suppurative pyelonephritis (surgical kidney).

Among the more important complications arising outside the urinary system must be

## PROSTATE, NEW GROWTHS OF

mentioned pronounced degenerative changes in the cardio-vascular system and, as the result of constant straining, hernias, with their attendant evils of strangulation, etc., hæmorrhoids, and complete prolapse of the rectum.

**Treatment.**—When slight vesical irritability is the only symptom, and the residual urine is clear and amounts to 2 oz. or less, no active treatment is required. Indeed, it is harmful in the early stages, and catheterization, except perhaps on one occasion if it is required as a diagnostic measure, should most studiously be avoided. Once infected, no amount of irrigation or other form of treatment short of operation will ever render the urine aseptic again in these cases. The patient should be warned against exposure to cold, and excess of alcohol. If possible, he should avoid placing himself in a position in which a call to micturate cannot be responded to without considerable delay.

In the more advanced cases, when increased frequency of micturition begins to interfere with the patient's social duties and rest at night, or the formerly slight obstructive symptoms have culminated in an attack of acute retention, more active measures are imperative. Two methods of treatment are then available.

(a) **Regular catheterization.**—The patient should be introduced into catheter life with every reasonable precaution. He should be provided with a suitable catheter—a firm rubber one (No. 12 E.) is the safest—and this, together with its accessories, oil, etc., should be boiled upon every occasion immediately before use. The hands and penis should be carefully washed and, if feasible, the patient should be instructed to wear rubber gloves. If, in spite of these precautions, an attack of cystitis occurs, it should be treated on the lines suggested under *CYSTITIS*.

(b) **Operation.**—A prostatic adenoma may be removed either by the suprapubic or the perineal route. In this country the suprapubic operation is the one almost universally adopted, and has no greater mortality than that performed by the perineal route. Unquestionably the drainage obtained by the former method is unequalled, and probably injury to the ejaculatory ducts is more likely to be avoided. Again, in perineal prostatectomy there is quite an appreciable risk of wounding the rectum or the compressor urethræ, and epididymitis is more commonly seen as a subsequent complication. Therefore, encapsuled adenomata should be

enucleated by the suprapubic method, and non-encapsuled tumours—in other words, the small “fibrous” prostate—treated by perineal prostatectomy or prostatotomy.

When there is a prominent intravesical projection, or when a pronounced soft enlargement can be made out from the rectum, it may be safely assumed that the tumour is of the encapsuled variety.

If, per rectum, the gland shows little or no enlargement, and if cystoscopy reveals only a slightly raised rim around the meatus, difficulties are likely to be met with. In a case of this description, if the prostate feels softer than the normal organ, it should be enucleated from above, but if, on the other hand, it gives a hard, firm, unyielding sensation to the examining finger, then the best result will be obtained by perineal prostatotomy.

**Prognosis.**—It cannot be denied that in the past men have completed happy and useful lives during the ten or fifteen years in which they were dependent upon the catheter, and have eventually died of some intercurrent malady. These cases must be regarded as exceptional. In most patients, indeed in practically all of the hospital class, catheter life invariably means the introduction of sepsis sooner or later.

Suprapubic enucleation, considering the age and general condition of the patients upon whom it is performed, is an operation of surprisingly low mortality.

In suitable cases the relief of the obstructive symptoms is almost perfect, and the general condition of the patient improves to a surprising degree.

With the advance of medical education, the time will come when operation will be recommended with confidence to these patients as the primary treatment, and not merely as a last resource to an old man worn out with years of septic absorption and loss of sleep.

A judicious selection of cases should be made, and prostatectomy urged in those instances only in which renal and cardio-vascular changes have not been allowed to progress to a dangerous degree. Operation is essential when one or more of the following conditions are present:—

1. Pain, hæmorrhage or obstruction, rendering regular catheterization impossible.

2. Repeated attacks of acute retention.

3. Residual urine of such an amount that even catheterization twice daily fails to make the patient comfortable.



## PROSTATE, NEW GROWTHS OF

4. Severe cystitis in which bladder drainage is required.
5. Vesical stone.

### B. CARCINOMA OF THE PROSTATE

**Pathology.**—Carcinoma of the prostate is of the spheroidal-celled variety, and is usually a slowly-growing tumour of the dense fibrous type, which may take three or four years to kill the patient. It usually arises in the unenlarged prostates of men of 60 or 70 years, but, exceptionally, may be seen in patients as young as 45. The main direction of spread is upwards and outwards into the vesiculæ and bladder base, but it rarely perforates the mucous membrane of the latter or invades the rectum until the latest stages. In some cases the primary focus may still be a small, hard nodule in the prostate, whilst a large inguinal mass of infected iliac and inguinal glands, or secondary growths in the bones or lungs, may already be in evidence; in others, the disease appears to be limited to the prostate itself for periods of many months and even years, and either metastasis appears very late, or the disease proves fatal from renal inadequacy before general dissemination can occur.

**Symptoms.**—The constant feature of malignant disease of the prostate is obstruction producing increased frequency and difficulty of micturition. Often the patients come under treatment for retention of urine, which, if once established, rarely intermits. Hæmaturia is unusual.

Pain is a variable symptom, and may range from a feeling of smarting deep in the perineum during micturition, or a sacral ache at night, to the most intense form of sciatica.

Per rectum, the prostate is usually somewhat enlarged and asymmetrical, giving an impression of stony hardness and absolute fixity to the examining finger. This change may be a uniform nodular one, or a single, hard, ill-defined plaque may be discovered in one portion of the gland only. With the extension of the growth upwards into the region of the bladder base the sharp definition of the concave upper border of the prostate is gradually lost.

**Treatment.**—Unfortunately, the great majority of these cases are only recognized, or come under observation for the first time, at a stage of the disease when any attempt at radical removal is impossible. In a few carefully selected cases extensive resection of the prostate complete in its sheath, the vesiculæ, and large portions of the bladder neck have

## PROSTATE, TUBERCULOSIS OF

been carried out successfully by a large perineal incision. The most that can usually be done for these patients is to render their last months of life more tolerable by establishing permanent suprapubic drainage. HAROLD W. WILSON.

### PROSTATE, TUBERCULOSIS OF.

**Etiology.**—Tuberculosis of the prostate gland seldom occurs as a primary lesion, but as a focus of disease secondary to tuberculosis of the epididymis or of the urinary organs. Previous gonorrhœal inflammation of the prostate may predispose to the deposition of tuberculous foci in the gland, but foci are often found in patients who have never had gonorrhœa. In some cases tuberculous disease of the prostate may be accompanied by a urethral discharge which may be mistaken for gonorrhœa, and only recognized as of prostatic origin in the later stages.

**Pathology.**—The tuberculous infection may reach the prostate by the blood-stream, or more commonly, when secondary to tuberculous disease in the epididymis, by the vas deferens or from the urethra.

The tuberculous process commences in the acini and in the prostatic ducts, the surrounding tissue becoming infiltrated, forming nodules which tend to caseate. The lobe only may be affected. The nodules break down, forming a cavity, or several may coalesce to form intercommunicating softened areas. These caseous areas may rupture into the urethra, into the base of the bladder, at the side of the trigone, or more rarely into the rectum.

**Symptoms.**—In the earlier stages of the disease there may be complete absence of symptoms, the nodules only being discovered on routine examination of the rectum in a case of congenital urinary tuberculosis. The onset of a urethral discharge without any venereal infection should lead to suspicion of a prostatic lesion, and a rectal examination should be made. Occasionally hæmaturia may be present when the focus ruptures into the bladder; it may be followed by pain and increased frequency of micturition. Hæmatospermia may also be present. The urine is at first clear, but when rupture into the bladder or urethra takes place the urine will contain pus and blood, and tubercle bacilli may sometimes be found. In the later stages, when the bladder becomes infected, there is frequent desire to micturate, pain along the urethra follows urination, and blood frequently appears in the last few drops of urine.

## PROSTATITIS

On rectal examination nodular areas will be felt in the prostate. The nodule is firm, well defined, and usually placed in the upper and outer portion of the gland, feeling as if embedded in the substance of the gland. When it has broken down and ruptured into the urethra or bladder a distinct cavity may be felt, the remaining lobe being the more prominent or containing unruptured nodules. In all cases careful examination should be made of the testes and epididymes as well as of the kidneys, ureters, and bladder, and of the lungs, joints, etc., to ascertain if any other tuberculous infection is present.

**Prognosis.**—In the early stages there is a tendency to cicatrization and recovery under suitable treatment, but recrudescence may occur after long periods of quiescence. If rupture takes place into the bladder, etc., tuberculous infection may spread, and the prognosis is less favourable, especially if there is also septic infection.

**Treatment.**—As a rule, primary treatment must be directed to disease in the genital or urinary system. For disease in the prostate, treatment on general lines should be carried out, and all local treatment to the prostatic urethra or the passage of instruments should be avoided. The most efficacious method of treatment is that by injections of tuberculin (T.R.), which should commence in doses of 500 mg., increasing weekly to 500 mg., and continued for months. At the same time the patient should be placed in the best hygienic conditions by residing at such places as Broadstairs or in Egypt, and a generous diet should be prescribed. Suppositories of belladonna  $\frac{1}{4}$  gr., 4 gr. of potassium iodide, or 2 gr. of ichthyol may be employed to relieve pain, and urinary disinfectants combined with sandalwood oil are useful.

Operative treatment on the prostate by erosion of the foci through a perineal incision is frequently followed by a persistent tuberculous fistula, but if pain in the late stages is severe a permanent suprapubic drainage of the bladder may give relief.

R. H. JOCELYN SWAN.

### PROSTATIC CALCULI (see URINARY CALCULI).

**PROSTATITIS.**—This article is concerned with *acute* prostatitis only, the chronic form, which is usually a complication of gonorrhoea, being sufficiently considered in the article on that subject.

**Etiology.**—Inflammation of the prostate is always due to bacterial infection. In the vast majority of cases the infecting agent is the gonococcus, which directly invades the gland from the posterior urethra. Prostatitis may also arise spontaneously as the result of a hæmatogenous infection, or follow the careless passage of a metal catheter or bougie in the case of stricture. It then usually occurs as part of a cysto-prostatitis, and is due to organisms such as *B. coli*, staphylococci, or streptococci.

**Symptoms.**—The onset is sudden, with a sharp rise in temperature and general malaise. There is much throbbing pain, increased on defæcation, felt deep in the perineum and rectum. Retention of urine may occur, but usually micturition is possible although exceedingly frequent and painful, and the urine may be blood-stained. Per rectum, there is exquisite tenderness and some fullness of the prostate, with oedema of the surrounding tissues.

**Prognosis.**—Under appropriate treatment a certain number of the cases undergo complete resolution within the course of two to three weeks, but usually the disease gradually passes into a chronic stage and an indurated and enlarged prostate may persist for many months. In a small percentage the disease progresses to suppuration.

**Treatment.**—Any local treatment of the urethra should at once be discontinued. The patient must be confined to bed on a light diet, and given large quantities of bland fluids; the bowels must be opened with calomel 5 gr. and saline aperients. Hot fomentations to the perineum and lower abdomen, or hip-baths, greatly relieve the pain, and should be combined with hot enemata and, if necessary, a suppository of morphia and belladonna.

A *prostatic abscess* is usually the result of an acute prostatitis. With the onset of suppuration the temperature, already high, rises still more and is often accompanied by rigors. The pain is greatly increased and radiates widely, and micturition becomes even more frequent and painful. Per rectum, the gland is greatly increased in size and conveys at first a boggy feeling to the examining finger, but later, as the pus approaches the surface, a softer and more elastic spot becomes evident. If left, the pus is usually discharged into the urethra, but may point in the perineum or, more rarely, rupture into the rectum.

**Treatment** consists in incision and drain-

## PRURIGO

age. The posterior surface of the prostate is exposed by a curved prerectal incision in the perineum, and the pus evacuated by piercing the prostatic sheath with a pointed pair of dressing forceps.

HAROLD W. WILSON.

**PRURIGO.**—The term prurigo should not be confused with that of pruritus. Pruritus means simply itching. Prurigo is the name given to an affection in which there is intense itching accompanied by a form of papular eruption. At the present day the use of the word prurigo is by many authorities confined to a severe and lifelong complaint, rare in this country, and known as *Hebra's prurigo*. Some observers, however, recognize a milder form of prurigo, which they call *prurigo mitis*. Others, perhaps the majority, merely use the term in a symptomatic sense to designate a particular form of papular eruption which may accompany long-continued pruritus from any cause, and they generally include under this term a circumscribed prurigo more generally spoken of as *lichenification*, the result of any form of local pruritus. Sometimes these conditions which result from long-continued pruritus and scratching are termed "pruriginous." But this is a word which most are agreed is better discarded, as leading to confusion.

It will simplify this subject to describe first of all the form known as Hebra's prurigo, and to discuss afterwards the milder forms, prurigo mitis and lichenification.

### PRURIGO OF HEBRA

This affection occurs most often in males of the poorer classes, and nearly all cases seen in London are in Polish Jews living in the East End. It usually appears within the first eighteen months of life, and lasts during the rest of the patient's existence. The characteristic features are an intense generalized pruritus, accompanied by an eruption of small pale-red rounded papules, chiefly upon the extensor surfaces of the limbs, the abdomen, and the buttocks. In course of time the whole of the skin becomes thickened and pigmented by chronic inflammation, and the lymphatic glands in the axillæ and groins are chronically enlarged. The constant scratching may lead to excoriations and crusting of the papules. The thickened skin becomes covered with striated excoriations, and the hairs rubbed away. There are periods of remission and of exacerbation, and during the latter urticarial wheals may be seen. The subjects of this

disease are generally poorly nourished, irritable and depressed, and suffer from insomnia. The causes of the complaint are unknown. It is intensified by neglect of hygiene and of treatment, and may be mitigated by good feeding and proper care, though it is probably incurable. Frequent baths and inunctions with any form of grease are the best treatment.

### PRURIGO MITIS AND LICHENIFICATION

In order to clear the ground, it may be stated as a well-known fact that any part of the skin may, as the result of long-continued scratching and rubbing, become the site of a chronic inflammatory condition which manifests itself in the form either of discrete papules mixed with pigmentation and excoriations, or of thickened dusky-red or reddish-brown patches or plaques called "lichenification." These two conditions have been called "prurigo mitis" and "circumscribed prurigo"; but they are now usually regarded as merely the secondary results of scratching and not as a primary condition, and it is generally recognized that they do not constitute a definite disease but that they may be met with in cases of long-continued pruritus from any cause. The more easily recognizable and more common of these is the so-called circumscribed prurigo, better known as lichenification.

**Lichenification** of the skin may be seen under various conditions: it occurs around the anus, on the perineum and inner sides of the buttocks as the result of scratching and rubbing due to pruritus ani; about the vulva and adjacent parts in pruritus vulvæ; at the bends of the elbows and behind the knees in cases of chronic eczema; at the nape of the neck as the result in the first instance of itching from a localized seborrhœa of the scalp, or in one or more oval patches on the forearms, thighs, legs below the knees, or other parts, as the result of itching set up in the first place by some trivial cause such as gnat-bite, or by a chronic eczema, by scabies, or indeed by any cause. The explanation of this lichenification is that, because of the itching, the patient scratches; the scratching sets up a local inflammation, which itself gives rise to further itching, until eventually the itching and scratching become a nervous reflex impossible to control, and the lichenification grows more and more pronounced. Clinically, this lichenification is seen as one or more oval patches of thickened and hardened skin, of dusky-red colour, with the surface shiny and "quadrillated," i.e. with the

## PRURITUS

natural surface groovings exaggerated, and sometimes excoriated by scratching. The patient is constantly scratching and rubbing this patch or patches, which accounts for their oval shape. Exactly the same conditions are observed, and are to be accounted for in the same way, when the lichenification occurs around the anus or vulva, or at the bends of the elbows or knees.

The **treatment** of lichenification is difficult. An endeavour should be made to prevent the rubbing and scratching which keeps it up, or in other words, to break the reflex habit; and the most efficacious method of doing this is by the application of X-rays. It is best to give to each affected part a full Sabouraud-pastille dose of X-rays and to repeat this once or twice at monthly intervals. Under this treatment the itching and scratching cease, and the area of lichenification becomes less infiltrated and finally disappears. Alternative methods are to paint the affected areas with a solution of silver nitrate 15 gr. in sp. ætheris nitrosi 1 oz. every few days, or to rub the part daily with liq. picis carb.

**Prurigo mitis.**—As has been said, in some cases of long-continued pruritus, from whatever cause, the skin becomes covered with small firm rounded papules, in addition to more localized areas of lichenification. By some authorities it is held that there exists apart a definite complaint, in persons of neurotic temperament, characterized by intense itching and eruption of prurigo papules and patches of lichenification, which may be regarded as a distinct affection and called prurigo mitis; but the majority of observers would probably regard the prurigo papules as a secondary condition, which may occur in any long-continued pruritus. The **treatment** of prurigo mitis is that of the pruritus and of the condition which gives rise to it, and is discussed under PRURITUS.

H. G. ADAMSON.

**PRURITUS.**—Itching is a symptom which may occur not only in many diseases of the skin, but also in connexion with many constitutional and visceral diseases. In certain skin affections it is often the most prominent feature, the main reason the patient has for seeking advice, and its cure the chief aim of our therapeutic efforts. In other instances it may furnish a clue to the discovery of some important general or visceral disease, and this fact should never be lost sight of in the presence of a case of pruritus.

From an etiological point of view cases of pruritus may be divided into four groups as follows:—

1. Those in which the itching is caused by the presence of animal parasites.
2. Those in which the itching is a symptom of some definite skin affection.
3. Where it results from abnormal constitutional conditions or from diseases of internal organs.
4. Where no local source of irritation can be discovered, and there is no skin affection and no evident disease of internal organs. Such cases have been called "mental pruritus" or "neurotic pruritus."

For clinical convenience pruritus is usually divided into two classes, viz., general pruritus and local pruritus.

**General pruritus.** In investigating a case of itching, one must first exclude local parasites, especially those of *pediculosis* and *scabies*. These are among the commonest causes of pruritus, and no station in life protects from them. In all cases of generalized itching this complaint should be first thought of, and the wrists and hands should be examined for scratch marks or for burrows of the acarus. In males the burrows may often be found upon the penis, and in children upon the feet. *Pediculosis corporis* (except under war conditions), is seen almost exclusively in elderly people, and especially in elderly alcoholics. Scratch marks about the shoulders should be looked for, and the punctate hæmorrhages produced by the mandibles of the louse. It may be difficult to discover the insect in the folds of the linen.

If a *skin eruption* is present, this may be the result of the scratching; for example, impetiginous or eczematous lesions may result from the scratching in scabies. In long-continued pruritus the skin may show papules, erythematous patches and scratch marks, and, after a time, more or less pigmentation. Of the skin eruptions in which pruritus is a prominent symptom the chief are eczema, lichen planus, and urticaria, and the less common but serious affections known as dermatitis herpetiformis and the pre-mycotic stage of mycosis fungoides; in the presence of a long-continued dry eczematous condition accompanied by pruritus it is important to bear in mind the question of mycosis fungoides.

Having satisfied one's self that the itching is not the result of animal parasites, and not due to a definite skin disease, investigations should

## PRURITUS .

then be made into the general condition of health and the state of the viscera.

In aged people one meets with what is called *senile pruritus*. It seldom occurs before the age of 65, and is usually associated with senile atrophy of the skin, though chronic alcoholism and pediculosis are often found to be the exciting causes.

In female patients pruritus may accompany some *disturbance of the sexual organs*. It is especially common at the climacteric, and may occur in connexion with pregnancy (*see Pruritus of Pregnancy*, under *VULVA, DISEASES OF*).

Pruritus may also depend upon more serious affections of the internal organs, such as *malignant disease* of the liver, of the gall-bladder, of the stomach, or of the uterus. It occurs as a symptom of *diabetes* or of *Bright's disease*, and in every case of persistent pruritus the urine must be examined for the presence of sugar or albumin. It is a frequent accompaniment of *jaundice*. It may be a symptom of *tuberculosis* or of *heart disease*.

There is a not very uncommon form of pruritus known as wintry pruritus (*pruritus hiemalis*) which occurs in some persons during the winter months. It may be general, but it affects particularly the external surfaces of the limbs, and especially the thighs, and it is generally worse on exposing the body to cold—that is, on undressing or getting out of bed. People who suffer from this form of pruritus are generally neurotic or gouty.

**Local pruritus.** The most important of these local forms of pruritus are pruritus ani and pruritus vulvæ.

Pruritus ani forms the subject of the next article, and need not be considered here. *Pruritus vulvæ* may depend upon a local cause, as vaginitis, threadworms, or irritating discharges from the cervix or uterus. It may arise also as a reflex from uterine or ovarian disease, and it may be due to pregnancy. The local irritation set up by diabetic urine is a cause which must not be forgotten, and, as in general pruritus, one should never omit to examine the urine for sugar.

To sum up, in investigating a case of pruritus unaccompanied by an obvious skin eruption, we should (1) first of all exclude scabies and pediculosis; (2) search for evidence of disturbance of the digestive functions; (3) examine the urine for sugar and for albumin; (4) examine the chest, heart, and abdomen for evidence of organic visceral disease; (5) when necessary examine the uterus and ovaries in a female patient; (6) search carefully for any

local trouble in pruritus ani and pruritus vulvæ.

**Treatment of pruritus.**—Whenever the pruritus is associated with a cutaneous disease, an important part of the treatment is to prevent the irritation of the skin by the scratching which the pruritus induces, since such irritation generally tends to aggravate the disease and to prevent its cure. Hence the great value of protective applications in eczema and of antipruritic applications in urticaria and in lichen planus or prurigo. Some writers advise the administration of narcotics, and in cases of infantile eczema, chloral may be given at bedtime in doses of 15–20 min. of the syrup to a child of six months, or half a drachm to a child of one to two years. In older persons bromides are better and safer than chloral. Opium should seldom be given, as it usually tends to aggravate the itching. Such drugs, however, are not often required if proper precautions be taken to protect the eczematous areas from irritation by exposure, by clothing, and by scratching.

In pruritus due to *scabies* appropriate treatment for the affection usually quickly gets rid of the itching, or if it persists after the cure of the scabies a mild tar lotion is effectual in removing it.

*Senile pruritus* is sometimes relieved or even cured by the avoidance of alcohol or by the removal of pediculi; and an excellent and cleanly method of treating pediculosis of the body is that of making the patient wear a piece of sulphur in a small muslin bag slung from the neck. Those cases of senile pruritus to which no such cause can be assigned are of much less favourable prognosis; many of them can be relieved by an empirical remedy, viz., cannabis indica, beginning with 5 min. of the tincture three times a day and increasing to 20 or 30 min. for each dose. It must be well diluted and taken after meals.

In pruritus of *diabetes*, of *albuminuria*, and of *cardiac disease*, these diseases must, of course, receive the first attention. Cardiac cases are often relieved by small doses of digitalis. *Winter pruritus* will often cease on stopping the cold morning bath and adopting warmer clothing. In many cases of pruritus cod-liver oil internally is useful as improving the general condition of the skin.

As empirical remedies in obstinate cases, one of the following drugs may be chosen, viz. quinine in large doses, antipyrin or phenacetin in doses of 10 gr., gradually increased.

## PRURITUS

In intractable forms of pruritus, injections of pilocarpine are often of great service. In senile pruritus especially, the injections may be continued for weeks or months. The most satisfactory method of employing the drug is as follows: The patient takes a warm bath in the evening just before going to bed; a hot drink is then given and an injection of pilocarpine  $\frac{1}{10}$ – $\frac{1}{8}$  gr.; profuse sweating occurs and the patient gets several hours' sleep, just at the time when the itching is most troublesome. This proceeding may be repeated nightly for weeks and sometimes leads to the permanent cure of the pruritus.

In all cases local applications are useful, and the list of such applications is almost endless. Among the most useful are those of the disinfecting type, such as liquor carbonis detergens, carbolic acid, and sanitas—of course, suitably diluted. To the lotion may be added such powders as zinc oxide or calamine. In many cases cold applications are found to relieve, such as sponging with vinegar and water, or with alcohol and ether, and afterwards powdering with starch powder. Lotions may most conveniently be applied by means of a spray. A lotion of perchloride of mercury,  $\frac{1}{4}$ –3 gr. to the ounce, is cleanly and odourless. Menthol 5–10 gr. in dilute alcohol 1 oz., is a useful lotion.

In *pruritus vulvæ* the rational treatment is, if possible, to remove the cause. It is important to insist upon local cleanliness. Relief may often be obtained by bathing the parts with a warm, freshly-made, saturated solution of boric acid and, after drying thoroughly, the free application of a simple zinc ointment, though of course here again local or reflex causes should be sought for and, if possible, relieved. We have now, too, in the X-rays a valuable remedy for obstinate cases of this form of pruritus. The application should be made according to the modern methods of dosage; a Sabouraud pastille dose being given once a month for two or three months. Most cases are immediately relieved by this treatment, and many remain permanently cured.

H. G. ADAMSON.

**PRURITUS ANI.**—A condition in which there is a constant and often most distressing itching sensation in the perianal region. It usually occurs in middle life or at a more advanced age.

**Etiology.**—Sometimes there is an obvious irritant such as external or internal piles,

## PRURITUS ANI

threadworms, fissure or fistula; occasionally the congestion caused by constipation may predispose to the condition, in other cases some constitutional dyscrasia may explain the symptom, in yet others it is excited by some article of diet, such as alcohol, coffee, red pepper or sugar, but often it is impossible to find any adequate cause.

**Symptomatology.**—The only symptom is severe itching around the anus. Examination shows an area of congested perianal skin with a thin patchy layer of white epithelium over it. The appearance is quite characteristic and is due to the excoriation of the skin from scratching and to the growth of epithelium over the raw area. The skin is often corrugated into folds radiating from the anus, and there may be raw patches.

**Treatment** should aim at removing the cause; it may be conducted on the following lines: (1) See that the bowels are kept freely open, and let the anal region be cleansed very carefully and gently but completely dried after each motion. (2) Remove any external tags of skin or external piles under local anaesthesia. (3) Treat any fissure or fistula or polypus by appropriate measures. (4) If the patient suffers from worms, treat accordingly. (5) Test the urine for albumin and sugar. In cases of diabetes or chronic Bright's disease give appropriate treatment. If the digestive organs are at fault, this irregularity should be corrected. (6) For local application to relieve the itching some of the following may be useful, viz.: alkaline sitz-baths (soda bicarbonate 2 oz. to 3 gallons of water); carbolic-acid lotion (1 in 20); ichthyol (10 to 50 per cent.); acetate-of-lead lotion (liquor plumbi subacet. 2 oz., distilled water to 8 oz.); calomel 1 dr. to 1 oz. of vaselin. Painting the affected area with solution of silver nitrate, 20 gr. to 1 oz., may occasionally relieve. Obstinate cases have been considerably improved by two or three exposures to the X-rays (suitably screened). Treatment by radium is sometimes beneficial.

Should these measures fail, division of the nerves going to the affected skin may be recommended.

ZACHARY COPE.

**PRURITUS OF PREGNANCY** (*see* VULVA, DISEASES OF).

**PRURITUS VULVÆ** (*see* PRURITUS).

**PRUSSIC ACID, POISONING BY** (*see* POISONS AND POISONING).

## PSEUDO-BULBAR PARALYSIS

**PSEUDO-ANGINA** (*see* ANGINA PECTORIS).

**PSEUDO-BULBAR PARALYSIS.**—A form of bulbar paralysis dependent upon lesions of the upper motor neurones connected with the bulbar cranial nerves; that is, of pyramidal fibres passing to the nuclei of these nerves in the brain-stem. The common cause is atheroma or syphilitic endarteritis of the cerebral vessels, giving rise to multiple small hæmorrhages and softenings, which are usually situated in the substance of the hemispheres, either in the white matter, the basal ganglia, or the internal capsules. These lesions are bilateral, but similar symptoms may be produced by a single lesion in the midbrain or pons, which interrupts the motor fibres at or near their decussation. Pseudo-bulbar symptoms are occasionally the result of a tumour in this region.

**Symptomatology.**—There is usually a history of two or more mild cerebral attacks followed by the onset of the symptoms. From the nature of the causative lesion, it follows that unilateral or bilateral hemiplegia is a common accompaniment of the condition. When bilateral this is usually slight on one side. The characteristic symptoms are dysarthria, dysphagia, difficulty in mastication and in phonation. The paralysis is one of voluntary movements only, and is combined with involuntary associated movements which constitute one of the most typical features of the disease. This is seen in the exaggerated action of the facial musculature in involuntary expressional movements, as in the spasmodic explosive weeping and laughter that occur.

The respiratory muscles also present this uncontrolled action during effort. The defects of movement are, therefore, compounded of weakness, spasticity, and involuntary overaction. There is great disturbance of articulation, which may result in complete anarthria in severe cases. The lips cannot be properly closed and therefore the labials *p*, *b*, *m* are uttered as *f*, *v*. The linguals are also imperfectly articulated. Owing to defective action of the soft palate the quality of the speech is nasal, and to these hindrances to normal articulation is added stuttering, owing to the explosive action of the musculature. Swallowing is also impaired; as a rule semi-solid pulpy food can be swallowed better than fluids or solids. The tongue and facial muscles are weak, and allow the escape of food and saliva from the lips. These defects may not be bilaterally symmetrical. From the multipli-

## PSEUDO-COXALGIA

city of lesions it follows that there may be aphasia, defects of vision and of sensation, and very commonly there are mental defects, such as apathy, dementia, confusion, and phases of excitement. Disturbances of the sphincters are not infrequent, but may be due to the impaired mental state.

**Diagnosis and prognosis.**—The disease must be differentiated from bulbar palsy, which is characterized by its gradually progressive course, associated with muscular wasting, fibrillation, and changes in the electrical reactions of the affected muscles, and with loss of both voluntary and involuntary movements (*see* MUSCULAR ATROPHY, PROGRESSIVE). Prognosis as regards improvement is bad. These patients may live for several years, during the course of which some improvement may occur. From the character and origin of the lesions it follows that none but symptomatic treatment is possible.

F. M. R. WALSHE.

**PSEUDO-COXALGIA.** (*syn.* Osteo-chondritis Deformans Juvenilis; Legg's Disease; Perthes' Disease).—A deformity in which there is alteration of structure of the upper epiphysis of the femur, causing limping and pain.

Pseudo-coxalgia has only been generally recognized as a common disease in recent years; previously, it was usually confused with tuberculosis of the hip.

**Etiology.**—The subjects are children between 3 and 12 years, more often boys than girls. The following causes have been suggested by different observers:—

1. *Error of development* of the whole of the upper epiphysis of the femur, affecting the head and trochanters; sometimes there are changes in the acetabulum.

2. *Trauma*, interfering with the limited blood supply of the head of the femur.

3. *Toxæmia or infection.*—In some of the more severe cases there is definite cachexia, and it has been suggested that the changes are due to congenital syphilis; in a certain number of cases the Wassermann reaction has been positive, in some there has been dental evidence suggestive of inherited syphilis (erosions and malformations of the first permanent molars and other units), and sometimes the condition has seemed to improve with specific treatment.

4. *Nutritional disease* of the muscles and bones.

The association of pseudo-coxalgia with congenital dislocation has occasionally been

## PSEUDO-COXALGIA

noted; sometimes its onset follows the reduction of the dislocation, or it may occur in the sound hip of a child suffering from congenital dislocation on the other side.

**Pathology.**—The pathological changes can be made out from the *X-ray appearances*. The disorder is most commonly unilateral, but both hips are affected in many cases; sometimes the symptoms are unilateral, but X-rays reveal more evident changes on the other side. The epiphysis of the head is flattened from above downwards, it is irregular in outline and density, and it may be segmented; there may be partial or total disappearance of the head, or it may be displaced downwards and produce coxa vara. The epiphyseal line is less distinct than normal, and may show irregular branchings; the juxta-epiphyseal region of the neck is irregular, with clear areas particularly in its proximal and lateral part; the lower and inner part of the neck is thickened, and sometimes the whole neck is stunted. The epiphysis of the great trochanter may be enlarged. Occasionally there is delay of development of the pelvis on the affected side, with irregularity of the acetabulum.

**Symptomatology.**—The patient is usually brought for treatment because there is a limp of acute onset; in most cases there is slight pain, but this is often quite absent. Sometimes there is cachexia. On examination, slight wasting of the muscles of thigh and buttock is apparent; the limb is in the normal position. There may be muscular spasm severely limiting all movements, but this disappears after a few days' rest; it is then found that abduction alone is definitely limited, though in most cases there is also slight limitation of flexion and extension, and of internal and external rotation; on forced flexion the limb becomes abducted. On palpation, owing to the wasting, the great trochanter appears unduly prominent, and slight thickening of the neck, especially on the inner side, may be detected. There is sometimes a little shortening, especially in older children, and in these cases there is scoliosis. Trendelenburg's sign is usually present; there is no pain on jarring the hip.

**Diagnosis.**—The disease has often been mistaken for early *tuberculous arthritis*, and in recent years many cases have been discovered in the special hospitals for hip disease. In tuberculosis, however, there are more wasting and pain, and more spasm and limitation of all movements, while the limb is held in the typical position of flexion, abduction, and external

## PSEUDO-SCLEROSIS

rotation. The X-ray appearances, fully described above, settle the diagnosis. In tuberculosis the rarefaction is more in the diaphysis, especially in the under aspect of the neck, and less in the head itself. In early tuberculosis the X-ray changes are slight and the symptoms are severe, whereas in pseudo-coxalgia the converse is the case.

**Prognosis.**—In most cases recovery occurs, even without treatment, but pain is relieved and cure is hastened by rest. As a rule complete recovery, as shown by X-rays, takes place within three years, but symptoms do not usually last more than one year. Sometimes a mushroom appearance of the head, simulating osteo-arthritis or coxa vara, persists, and occasionally permanent slight shortening of about half an inch results and causes scoliosis, which is liable to progress during adolescence.

**Treatment.**—When there is much spasm and pain the child should be kept in bed, extension being applied with the hip in the abducted position. If there is little general spasm, but pronounced adduction, this should be overcome under general anaesthesia, and the limb fixed by a plaster-of-paris spica; the child should be kept from standing or walking for three months. In the later stages, and in the less severe types, the child is allowed to walk with a Thomas calliper splint, or with the apparatus devised by Gauvain; this consists essentially of bands passing round the pelvis and the condyles of the femur, with an adjustable and extensible posterior rod that transmits the weight and deflects it from the hip.

C. W. GORDON BRYAN.

**PSEUDO-GLIOMA** (*see* VITREOUS, AFFECTIONS OF).

**PSEUDO-HYPERTROPHIC PARALYSIS** (*see* MYOPATHY).

**PSEUDO-LEUKÆMIA** (*see* Diagnosis, under LYMPHADENOMA)

**PSEUDO-PARALYTIC MYASTHENIA GRAVIS** (*see* MYASTHENIA GRAVIS).

**PSEUDO-SCLEROSIS.**—This term was coined originally by Westphal to describe certain cases which bore a close clinical resemblance to disseminated sclerosis, but in which the pathological lesions of the latter disease were not found. Under this title a number of cases have been grouped which there is little reason to doubt belong to differing classes of nervous disease, and it is also fairly certain



## PSORIASIS

that incompleteness of pathological examination is the explanation of not a few cases being thus described. The symptoms, as observed originally, were mainly tremors or other involuntary movements, stiffness of the skeletal musculature, slowness of voluntary movement and of speech, psychical disturbances, and epileptiform convulsions.

Recently certain cases have been grouped in this ill-defined category in which the symptoms were as enumerated above, and in which a fairly close resemblance to the disease known as progressive lenticular degeneration was observable. These cases, moreover, were discovered post mortem to have cirrhosis of the liver, but the characteristic lenticular changes were wanting; in their place was found a generalized neuroglial overgrowth involving more particularly the corpus striatum, optic thalamus, subthalamic region, pons and nucleus dentatus of the cerebellum. The exact relation of this sub-group of pseudo-sclerosis to progressive lenticular degeneration is not clear. It may be mentioned that, clinically, some cases in this sub-group showed a curious pigmentation of the cornea in the shape of a ring round its periphery.

Obviously the subject is one on which further research is desirable before any adequate treatment can be evolved.

S. A. KINNIER WILSON.

**PSILOISIS** (*see* SPRUE).

**PSOAS ABSOESS** (*see* SPINAL CARIES).

**PSORIASIS.**—A chronic inflammatory disease characterized by red scaly macules or slightly raised papules which spread to form circumscribed red patches covered with silvery scales.

**Etiology.**—Psoriasis is one of the commonest cutaneous affections in this country, constituting between 6 and 7 per cent. of all the cases of skin disease. It generally begins between the ages of 5 and 15, in rare instances may occur in infancy or early childhood, and occasionally does not appear till old age. It is equally common in the two sexes and in all classes of society. Though it may occur in any climate, it is most frequent in cold latitudes. Heredity is known to play a part in its etiology, for it may attack several members of a family, and in a large proportion of the cases there is a history of it in one or other parent. The relation of the disease to disturbances of the general health is doubtful. It has been described as "a disease

of the healthy," still it not infrequently occurs in association with some general disorder, such as rheumatism, gout, or neurasthenia, and numerous instances are on record where relapses were the result of some serious illness or nervous disturbance. Season appears to have some influence on its inception and recurrence, as it is liable to commence in spring and to recur towards the end of summer. Traumatism may be a predisposing factor, as the earliest sign of the disease has been known to occur at the site of an injury or on a vaccination scar, while pressure and friction are liable to determine the position of new lesions.

**Pathogenesis.**—The true nature of the disease is uncertain. By some it is thought to be the result of a peculiar diathesis due to a deranged state of the blood and the circulation in it of some toxin; others consider it due to some error in metabolism such as might be caused by excessive meat-eating, but elaborate experiments to prove this contention have given negative results; others, again, believe it to be parasitic and caused by a microbe, either inoculated locally or reaching the skin via the bloodstream, but, though various micro-organisms have been grown from the scales, none has been proved specific.

**Symptomatology.**—The characteristic essential lesion is a reddish, slightly raised, rounded papule, circular in outline, varying in size from a pin's head to a lentil, and covered with a fine whitish scale which on being gently scratched assumes a distinctly silvery appearance. When this scale is removed a red shiny film is left dotted over with minute bleeding-points. The lesions spread peripherally to form scaly plaques which may reach the size of a crown-piece or larger, and may coalesce to form irregular figures. The early lesions are flat and scarcely perceptible to the touch, while the older patches may be slightly raised, indurated, and rough. When the disease is actively spreading the patches are bright red in tint but later become dull pinkish. In plethoric and alcoholic people they may have a brownish tinge, while in dependent parts like the legs they may be livid. When they reach about the size of a crown-piece they usually cease to grow, and either fade all over, or in the centre, forming a ring which in turn disappears, leaving no trace, or at most a slight pigmentation.

Though all the cases show the same fundamental lesions, no two cases are identical in distribution and type of eruption. It is customary to apply distinctive names to the different



PLATE 26.—GUTTATE PSORIASIS, WITH TYPICAL LESIONS OVER ELBOW.



**PLATE 27.—PSORIASIS, WITH CIRCINATE SCALY  
PATCHES.**

## PSORIASIS

types, such as *punctate psoriasis* when the lesions are small and usually follicular, *guttate psoriasis* when they are like drops of mortar stuck on the skin (Plate 26), *nummular psoriasis* when they are the size of a small silver coin, *annular* or *circinate psoriasis* when they are ringed (Plate 27), *gyrate psoriasis* when ringed lesions have coalesced to form gyrate figures, *diffuse psoriasis* when large areas are involved, *rupeoid psoriasis* when the scales are limpet-shaped. But these differences in type are of secondary importance and dependent on peculiarities of the skin of the individual affected.

The most common sites for the eruption are the knees, elbows, scalp, extensor aspects of the limbs, and lumbar region, but it may affect any part of the skin, being least common on the face, palms, and soles. In distribution it tends to be symmetrical, and may be localized to a few areas such as the elbows and knees or widely disseminated over the cutaneous surface. It may attack the nails, usually producing a thickening of the nail-plate, with white opacities and small pits.

Subjective symptoms vary in different cases; they may be slight or wholly absent, or there may be more or less itching where large areas are involved, especially in nervous or gouty subjects.

The disease as a rule does not involute spontaneously, though it may do so, and if left alone is apt to be steadily progressive up to a certain point and then to remain stationary. Under suitable treatment an attack will usually clear up in a month or six weeks, but it tends to recur sooner or later.

In some cases the typical lesions of psoriasis may be complicated by thickening or lichenification from rubbing, or by eczematization or pustular dermatitis from scratching and the secondary inoculation of pus cocci, or the disease may be transformed into exfoliative dermatitis by excessive treatment. On the other hand, a symbiosis of psoriasis with other cutaneous affections may occur, and it may be met with in association with syphilis or tuberculosis, or a singularly resistant psoriasis patch may be superseded by an epithelioma.

**Histopathology.**—Sections of an early lesion show a proliferation and down-growth of the prickle-cell layer of the epidermis, with oedema of the cells and dilatation of the intercellular lymphatic spaces. The process of cornification is interfered with so that imperfect horn-cells are formed, which are collected in

squames separated by air, causing the characteristic silvery appearance of the scale, and by the debris of broken-down leucocytes and epidermal cells. In the corium the superficial capillaries are widely dilated, the tissue around them is more or less oedematous, and there is a varying degree of perivascular cellular infiltration.

**Diagnosis.**—Psoriasis has to be distinguished from seborrhœic dermatitis, eczema, and syphilis. In *seborrhœic dermatitis* the essential lesions are yellowish-pink, irregular, ill-defined, covered with greasy yellowish scales, and distributed chiefly on the scalp, sternal and interscapular regions, axillæ, and groins, and in the scales certain characteristic micro-organisms known as the spores of *Malassez* can be detected. In *eczema* the patches are less clearly defined, the colour less vivid, the scaliness neither silvery nor so profuse, and in many cases there is definite "weeping." In the scaly papular *syphilide*, with which psoriasis is most liable to be confused, the scales are more delicate, of a dirty whitish tint, and when scratched off are followed by bleeding; the lesions are yellowish-brown, definitely indurated, leave a brownish stain when pressed by a piece of glass, are distributed more irregularly over the cutaneous surface, tend to affect the face and flexor aspects of the limbs, are usually associated with other symptoms of syphilis, and a positive Wassermann reaction is almost invariably obtained.

**Treatment. General measures.**—In severe cases, where the disease is widely distributed, rest in bed should be advised while the active treatment is being carried out, an ordinary light nutritious diet prescribed, and rich food, alcohol, and coffee prohibited. When there is much tension and irritability of the skin small doses of the wine of antimony give relief. Any definite derangement of the general health must be dealt with on general medical principles.

There are several drugs which are believed to have a specific action on psoriasis, such as arsenic, salicin, and thyroid extract. *Arsenic* is usually prescribed in the form of Fowler's solution, beginning with doses of 3 min. t.d.s., increased to the limit of toleration, continued for several weeks, and then gradually diminished. It should be given well-diluted with water after meals, and is contraindicated where there is indigestion or other derangement of the alimentary tract. It is uncertain in its action, and would seem to be of most value in first attacks in children. In acute cases in which the disease is widely distributed, *salicin* has been

## PSORIASIS

strongly recommended, in full doses of 15 to 20 gr. t.d.s. after meals, and is specially useful where the psoriasis is associated with rheumatic symptoms. *Thyroid extract* was once extensively employed in psoriasis, but has now fallen almost completely into disuse, as its action is even more uncertain than that of arsenic.

**Local measures.**—The special indications in the local treatment consist in the removal of the scales, the reduction of the lesions, and the after-treatment.

**Removal of the scales.**—When the eruption is extensive a daily warm bath with the addition of  $\frac{1}{4}$  lb. of bicarbonate of soda to 30 gallons of water should be given, the patient being immersed in the bath for 20 minutes and the scales rubbed off with soft soap. When the skin is irritable an emollient bath containing 2 lb. of starch or bran should be substituted. After the bath each patch should be rubbed with an ointment composed of salicylic acid 15 gr., glycerin of starch 2 dr., soft paraffin to 1 oz. When the scales are present on the scalp, it should be shampooed with soap liniment (green soap 2 parts, rectified spirit 1 part), an ointment containing salicylic acid 10 gr., ammoniated mercury 15 gr., and soft paraffin to 1 oz., being subsequently applied. When possible the hair should be cut short.

**Reduction of the lesions** is effected by means of one of the so-called reducing agents, of which the most effective are chrysarobin and tar. *Chrysarobin* is best used in the form of a 6-per-cent. ointment in vaselin rubbed into the patches daily after the bath. This causes a blanching of the patches and a reddening of the surrounding skin in about a week, and some time later a reddening of the patches themselves. The treatment should be continued until the patches and the immediately surrounding skin are uniformly red, when it should be stopped. A cleaner though less effective method of employing chrysarobin is to dissolve it in traumaticin (gutta-percha 1 dr., chloroform to 1 oz.) and to paint it on with a brush. Chrysarobin is the most effective of the local applications, but requires to be used with care and judgment. It is an irritating remedy, and if allowed to reach the eyes may cause a severe conjunctivitis, consequently it is better not to employ it about the face or scalp; it is dirty, staining the hair yellow, the skin a brownish-purple, and the clothing a brownish-yellow tinge which becomes purple on washing with soap; and if employed too actively it may

## PSYCHASTHENIA

transform the psoriasis into an exfoliative dermatitis. It is most suitable in cases with extensive patches where the inflammation is slight and the disease has reached its height, and in chronic resistant patches, and should not be employed when the eruption is acute and actively spreading. *Tar*, though less powerful than chrysarobin, is useful for patients with multiple lesions and sensitive skins. It is most frequently used in the form of the oil of cade, the solution of coal tar, or creolin. When the disease is widely distributed the patient should be immersed daily in a warm tar bath containing 2 oz. of the official solution of coal tar to 30 gallons of water, and after the bath the individual patches should be rubbed with an ointment containing oil of cade 2 dr., soft paraffin to 1 oz.

When the disease is limited to a few chronic resistant patches these can usually be removed by the X-rays, a three-quarter pastille dose being given, followed by a second in a fortnight if necessary. But the disease tends to recur as readily after X-ray treatment as after chrysarobin or tar, unless the exposures have been excessive.

In psoriasis of the scalp, after removal of the scales an ointment consisting of solution of coal tar, ammoniated mercury, and salicylic acid, of each 10 gr., white paraffin to 1 oz., should be rubbed in daily.

Nails affected by psoriasis should be softened by a 5 per cent. solution of caustic potash, scraped with a piece of broken glass, and an ointment containing salicylic acid 15 gr., soft paraffin to 1 oz., rubbed over them and pushed up beneath them.

**After-treatment.**—In a favourable case suitably treated the eruption should be got rid of in a month or six weeks, but to stave off a recurrence a modified treatment should be continued for several months, such as the following: The patient should have a warm bath three times a week and the skin be washed with ordinary toilet soap; after the bath the skin should be dried and anointed with olive oil, and a 2 per cent. beta-naphthol ointment should be rubbed over the sites of predilection every second night. J. M. H. MACLEOD.

**PSYCHASTHENIA.**—A functional nervous disorder, characterized by inability to regulate ideas and actions in a logical manner, together with difficulty in concentration and, in more severe cases, obsessions and emotional crises.

**Etiology.**—The affection is most common in

## PSYCHASTHENIA

subjects with a neuropathic inheritance, and generally shows itself in a mild form from childhood. It is most frequent among the educated classes, and is often associated with the so-called artistic temperament. Definite psychasthenic symptoms are likely to develop when a predisposed individual is exposed to emotional strain. Mental and physical overwork do not of themselves cause psychasthenia, but the neurasthenic condition to which they give rise makes the patient more liable to develop psychasthenia.

**Pathogenesis.**—Psychasthenia is a pure psychoneurosis, and has no organic basis of any kind. It is the result of keeping painful memories or conflicting instincts in the subliminal consciousness by more or less voluntary repression. The primary psychical symptoms may give rise to secondary physical phenomena owing to the influence of the emotions on sympathetic nervous activity, but the hypersecretion of the suprarenal and thyroid glands, together with the circulatory, digestive and other symptoms which result, are not an essential part of the condition.

**Symptomatology.**—A psychasthenic person finds it difficult to adapt himself to the realities of life; he hesitates when called upon to decide between two possible lines of action, and when at last he has adopted one he is full of doubts as to whether he has not made a mistake. He shows a want of confidence in all his doings. Though often unaware of the fact, his mental energy is largely taken up in repressing painful thoughts and conflicts, which are kept in the background of his mind in order to avoid distress. As a result of this his power of concentration is deficient, and an abnormal effort is required to recall past events. When in the daytime his mind is not fully occupied, the repressed thoughts and conflicts may give rise to emotional crises—attacks of apparently causeless emotion, such as dread, horror or terror, with their physical accompaniments of tremor, tachycardia, pallor, sweating, and even diarrhoea, or of laughter or weeping, popularly called “hysterics.”

Owing to the need of active thought to keep the distressing memories and mental conflicts buried, the patient often finds it difficult to fall asleep. When at last he sleeps, the controlling influence over his thoughts is relaxed, and repressed memories are likely to come into consciousness in a distorted form as nightmares, with the result that he wakes in a state of terror, trembling and bathed in sweat, but

often unaware of the cause of his emotion. The disturbed nights lead to exhaustion and secondary neurasthenia, with headache and tendency to mental and bodily fatigue; it is then often impossible to disentangle the neurasthenic from the psychasthenic elements of the composite clinical picture which the patient presents. The psychasthenic subject is often abnormally suggestible, and one or more of the physical expressions of his emotions, such as terror, stammering, or vomiting, may be perpetuated as an hysterical symptom.

In severe cases obsessions develop, an obsession being an inadequate idea or unsubstantial fear, commonly called a phobia, which intrudes itself into the consciousness in an irresistible manner without completely filling or dominating it. The classification of obsessions and the naming of phobias—agorophobia, claustrophobia, anthropophobia, and so on—is of no great importance: the essential thing to realize is that in every case, if the mental history of the patient is investigated with sufficient care, the explanation can be discovered why a particular obsession or phobia has developed.

**Prognosis.**—The prognosis depends upon the duration of the symptoms, and especially upon whether the individual was otherwise normal when the circumstances arose which gave rise to his psychasthenia. With skilled psychotherapy every case should improve, and in recent cases a complete cure can always be expected. When, as is frequently the case, the patient has always been of a nervous disposition, the outlook is less good, as although the symptoms may be greatly relieved, there often remains a tendency to relapse with a return of mental strain. It is quite impossible to say beforehand how long it will take to effect a cure. In rare instances a single psychotherapeutic conversation may be all that is necessary, even in an apparently severe case, whilst occasionally three months or more may be required for what at first appeared to be a mild and straightforward case. It is therefore of the greatest importance to offer no opinion as to the exact duration of treatment, as the suggestion that three months will be required is likely to result in the recovery, which might otherwise have taken place within a week, being considerably delayed.

**Treatment.**—The treatment of psychasthenia should be entirely mental; the old-fashioned rest cure and the administration of drugs are useless and often harmful. In

## PSYCHASTHENIA .

severe cases, however, it is essential to take the patient away from his home surroundings, but complete isolation is rarely required. It is most important to gain the patient's complete confidence, so that he becomes willing to speak about his most intimate thoughts and discuss affairs which he has kept hidden from his nearest relatives and most intimate friends. He is then encouraged to search his memory for the real origin of his symptoms. An investigation into his thoughts when he lies awake at night and into his dreams is often of great value. He is then led to face bravely the memories and conflicts he has been repressing. He is helped to solve his difficulties, and he is made to realize that a free discussion of the thoughts he has been attempting to repress, but which have prevented him from sleeping at night or have appeared in a distorted form as dreams, will produce the relief he desires. It is very remarkable how rapidly persistent nightmares, longstanding phobias, hitherto inexplicable emotional crises and other psychasthenic symptoms disappear as soon as the patient thoroughly understands the mental processes which have given rise to them. The analysis of his mental state might appropriately be called psycho-analysis, were it not for the fact that the term has unfortunately become attached to the special teaching of Freud, who believes that the suppressed psychical origin of the condition is invariably sexual. However right this may be as far as his Viennese practice is concerned, it is quite certain that the majority of cases of psychasthenia in England have absolutely no sexual element in them, although sexual psychasthenia is, of course, a well-recognized condition. Perhaps the term *psychological analysis* might be employed.

When the cause of the symptoms has been removed, the patient should be given mental exercises in order to restore his memory and powers of concentration. These quickly return, as he can now avail himself of the mental energy previously used up in futile efforts connected with his worries. A. F. HURST.

**PSYCHO-ANALYSIS** (see PSYCHASTHENIA).

**PSYCHONEUROSES** (see NEURASTHENIA ; PSYCHOTHERAPY).

**PSYCHOSES** (see PSYCHOTHERAPY).

**PSYCHOSES, EXHAUSTION, INFECTIVE, TRAUMATIC** (see CONFUSIONAL INSANITY).

## PSYCHOTHERAPY

**PSYCHOTHERAPY.**—This may be simply defined as the treatment of disorder by psychological means. Under the term are included all therapeutic methods which endeavour to influence the mental processes of the patient, with the object of producing thereby certain desired effects in the mental or physical organism.

**Sphere of application.**—In order to determine in what conditions psychotherapy may suitably be employed, it is necessary to consider certain general principles. Every clinical condition with which we have to deal may be regarded as the end-result of a network of causes whose action and interaction have produced the picture before us. In this network both physical and mental factors may play a part, but their relative importance varies greatly in different cases. In organic cardiac disease, for example, the various signs and symptoms presented to us may owe their origin in some degree to mental factors—emotional disturbances and so forth—but the part played by these causes is of little significance in comparison with that due to the physical factor present, the actual organic lesion. In a case of hysterical paraplegia, on the other hand, although physical conditions may have contributed to the result, the dominating factors in the chain of causation are purely mental. Now it is clear that, as therapeutics is necessarily dependent upon etiology, disorders for which physical factors are mainly responsible must be dealt with by physical methods of treatment, and disorders for which mental factors are mainly responsible must be dealt with by psychological methods of treatment. The sphere of application of psychotherapy can, therefore, be easily defined. It is mainly limited to those disorders in whose causation mental factors have played the chief part, disorders which are for this reason termed psychogenic. In other disorders it can only be employed as a subsidiary weapon with the object of removing those minor disturbances of mental origin which are often grafted upon organic disease. Hence, in order to determine whether and to what extent psychotherapy is applicable in any given case, it is necessary to determine how far mental causes are responsible for the disorder present. The result of this investigation will immediately furnish the criterion required. It is important to remember that the question at issue is not whether mental symptoms predominate in the clinical picture, but whether mental factors

## PSYCHOTHERAPY

predominate in the chain of causation which is responsible for this picture. In hysterical paraplegia, for example, the symptoms and signs are mainly physical, but the causes are almost entirely mental, and such a case is therefore eminently suitable for psychotherapeutic methods.

In the light of these considerations we may now pass on to enumerate the **disorders in which psychotherapy is applicable**. These are :

1. **The psychoneuroses.**—Under this term are grouped the various conditions known formerly as “functional nervous disorders,” and including hysteria, neurasthenia, psychasthenia, anxiety neuroses, obsessional neuroses, and at any rate the majority of the disorders called in the War “shell-shock.” In all this group, mental factors play a predominant part in the causation, and it is the field in which psychotherapy has its greatest utility and success.

2. **The psychoses.**—This term is used to designate the actual insanities. The extent to which psychotherapy is applicable here is at present very uncertain, the uncertainty being correlated with the fact that the relative part played by physical and by mental factors in the causation of these disorders has not yet been determined, and is a subject of dispute between conflicting schools of thought. There can be no doubt that mental factors exert an exceedingly important influence in fashioning the clinical picture presented by the patient, but whether they are primarily responsible for the disorder, or merely mould into particular shapes symptoms due to organic disturbances which have no psychic antecedents, is still an unsolved problem. The answer will probably prove to be very different in the different forms of insanity. In general paralysis, for example, the fact that the patient exhibits a certain delusion may be explained by the various psychological causes operative in his mind, but that mental disturbance exists at all is dependent upon the underlying physical disease. In other mental disorders, however, it seems possible that psychical factors may predominate in the causation, and if this should be established it is clear that psychotherapy ought ultimately to be capable of dealing with them. Whatever its future prospects may be, however, the present practical value of psychotherapy in the insanities is limited. The disorders in which it would seem to have its most likely field of application are dementia præcox and manic-depressive insanity, and certain observers

claim to have achieved promising results in their treatment by these methods. Even at the present time, however, psychotherapy is unquestionably of great value in the treatment of the insanities as an adjuvant measure, and much may be done by its means in prophylaxis and in the handling of early and incipient cases.

3. **General medicine.**—In every disease the clinical picture presented by the patient is coloured to some extent by psychical factors, and such factors also exert an influence, although a minor one, on the course and ultimate outcome of the disease. It is well recognized, for example, that the personality of the physician, and his ability to induce an atmosphere of cheerfulness and hope, play some part in treatment. Similarly, the authoritative position which the physician occupies lends to all his instructions and advice a powerful suggestive force, which is in itself capable of producing considerable modifications in the patient's condition. These instruments are inevitably employed, consciously or unconsciously, by every practitioner, and it is clearly desirable to employ them deliberately and with a due appreciation of their action, rather than blindly or with only the uncertain light afforded by common sense. Such a systematic use presupposes, of course, a reasonable acquaintance with the principles and methods of psychotherapy.

**Methods.**—A superficial perusal of the extensive literature that has appeared in recent years on the subject of psychotherapy would suggest that its methods are legion, and that almost every authority advocates some one method to the exclusion of all others. This diversity, however, except in so far as it relates to matters of detail, is more apparent than real, and it is possible to reduce the multiplicity of methods to the employment of four basic processes—*suggestion, persuasion, analysis, and re-education*. Although certain authorities rely mainly on one or other of these processes, the greater number are accustomed to make use of at least two and generally of all four. In my opinion all four have their place in treatment, but the method to be employed must be carefully selected to suit the individual case. The common misapprehension that suggestion and psychotherapy are practically synonymous terms is, of course, absolutely erroneous. Suggestion constitutes only one of the weapons of psychotherapy, and probably by no means the most widely applicable or efficient one.

1. **Suggestion** has been defined as “a process



## PSYCHOTHERAPY

of communication resulting in the acceptance with conviction of the communicated proposition in the absence of logically adequate grounds for its acceptance" (McDougall). Its therapeutic effect is dependent upon the fact that the belief thus induced in the mind of the patient is capable of modifying mental factors which are responsible for his symptoms, and thereby leading to the amelioration or disappearance of those symptoms. When, for example, an hysterical paralysis of the arm is treated by suggestion, the object aimed at is the introduction into the patient's mind of a firm conviction that the arm is no longer paralyzed and is capable of movement. If this is achieved the conviction counteracts the effect of the purely mental factors responsible for the symptom, and the paralysis disappears.

Although there are a great number of methods of applying suggestion, it must clearly be understood that in every instance the essence of the procedure is to implant in the patient's mind a conviction of the kind described, and the different methods only vary in the nature of the "vehicle," as it may be called, employed to facilitate this implantation. These various methods of employing suggestion may be divided into two main groups, *hypnotic* and *non-hypnotic*.

*Hypnotism* consists in the induction of a peculiar mental state whose essential character, so far as therapeutics is concerned, lies in the fact that it is accompanied by a high degree of suggestibility. That is to say, suggestions are accepted in the hypnotic state far more readily than in the normal state. It is possible to ensure, moreover, that the suggestion continues to exercise its effect after the patient has awakened, and thereby to achieve a more or less permanent removal of symptoms. Hypnosis may be induced by various procedures, for details of which reference should be made to the special works on the subject. When a suitable degree of hypnosis has been attained the physician gives curative suggestions to the patient; he suggests, for example, that an hysterical paralysis of the arm has disappeared, that the patient is now able to move his arm without difficulty, and will continue to be able to do so. If the procedure is successful the symptoms attacked do in fact disappear, at least temporarily, and sometimes permanently.

The advantages of hypnotism are that it is the most potent vehicle for the administration of suggestion available to us, that it is therefore often possible to obtain results by its use when

other methods of suggestion fail, and that its effects can be produced more rapidly than by other suggestive processes. Its disadvantages are partly those inherent in the employment of all methods of suggestion, which will be dealt with in a subsequent paragraph, partly those belonging peculiarly to itself. Although the latter are, of course, frequently exaggerated to a ludicrous extent, it would seem that the repeated use of hypnosis does tend to produce in the patient an undesirable dependence on the physician. Nevertheless, I am of opinion that hypnosis is a valuable psychotherapeutic weapon, provided that the cases for its employment are carefully selected, and that the treatment is carried out only by physicians experienced therein.

The *non-hypnotic* methods of applying suggestion may be further divided into frank or open suggestion, and suggestion by the aid of some physical vehicle. The former consists in conveying the desired suggestion to the patient in the waking state, and simply by word of mouth. He is told, for example, that a certain symptom will disappear, and, if the personality and authority of the physician are sufficiently impressive, the conviction will be instilled into the patient's mind and will produce its effect just as in the case of hypnosis. It is important to realize that everything the physician says or implies may exercise an action of this kind, and that it is therefore essential to guard against giving suggestions which may exercise a harmful effect. This last point cannot be too much emphasized, because it is unfortunately a fact that many neurotic conditions are aggravated and fixed by injudicious advice and treatment. A patient with a functional dyspepsia, for example, is subjected to repeated examination, the probability of ulcer or even malignant disease is openly discussed or implied by the physician's countenance, a course of careful dieting is prescribed, and various drugs are administered. The result is that the patient's previous apprehension that an organic disease is present is confirmed, the mental factors responsible for the condition are strengthened, and the difficulty of subsequent suitable treatment is greatly increased.

Suggestion by the aid of a physical vehicle includes all procedures in which a physical treatment is employed with the deliberate object of inducing thereby a suggestive effect. That is to say, the physical agent is used, not for any specific action which it may happen to possess, but simply as a means of inducing the

## PSYCHOTHERAPY

patient to accept a certain suggestion. The physical agent most frequently relied upon for this purpose is electricity, but drugs, baths, massage, etc., may similarly be employed. A patient with hysterical paralysis of the arm, for example, is told that directly the electric current passes through his arm he will recover the use of it. The current is then applied and, if the suggestion is successful, the paralysis disappears. It is essential, of course, to remember that the suggestion is the cardinal factor in the treatment, and that if the electricity is applied without at the same time inducing in the patient's mind the conviction that a cure will result, no effect is likely to be produced. Drugs, massage, and hydrotherapy may be used in the same way, and every practitioner should estimate, when considering the effect apparently produced by the medicines he prescribes, how much of this is to be ascribed to suggestion.

The applicability of suggestion as a curative agent is severely limited, and it is necessary to understand on what its limitations depend, in order that only suitable cases may be selected for its application. The essential limitation is often expressed by saying that it removes symptoms rather than causes. To put the matter more accurately, a psychoneurosis is the result of a network of interacting causes whose final effect in certain cases is to produce in the patient's mind the conviction that certain disturbances or symptoms are present, and upon this conviction the actual symptoms are immediately dependent. The action of treatment by suggestion in such cases as these is to remove the conviction in question and to substitute for it the opposite conviction, namely that the symptoms have disappeared or are about to disappear. Suggestion therefore achieves its end by breaking the chain of causation at its final link, the primary causes being left *in statu quo*. It follows from this that suggestion is only applicable to cases where a conviction of the kind described is present, and that even here it can rarely by itself produce a radical cure, because the primary causes may easily be lighted up again and lead to a relapse.

Applying these considerations to the conditions met with in actual practice, it may be said that the main field for the application of suggestion is to be found in hysteria, though even here it is only satisfactory provided that other measures are taken to deal with the more essential causes responsible for the disorder.

It is also suitable for those functional disturbances of the digestive and other organs which are dependent upon preoccupations in the patient's mind, though it is inferior in these cases to persuasion and re-education. It is sometimes, though rarely, useful as a palliative measure in obsessional neuroses. Lastly, it is a valuable accessory agent in the course of other methods of treatment, especially for the recovery of amnesias, for the rearrangement and modification of causal factors elicited by analysis, and occasionally for dealing with such symptoms as insomnia.

2. **Persuasion** may be defined as a process whereby the mental factors responsible for a functional disorder are modified by the use of logical arguments. The method of application is to explain to the patient the nature of the condition from which he suffers and the fact that it is not dependent upon organic disease, and to demonstrate to him that the capacities he believes he has lost are actually still present. To take once again the example of hysterical paralysis of the arm, the patient is shown that the signs of an organic paralysis are absent, it is explained to him that the loss of power is due merely to a temporary loss of control, and the actual persistence of the capacity for movement is demonstrated by, for instance, supporting the arm for a time and then removing the support while the patient's attention is diverted. In all probability the arm will preserve its position for a time sufficient for the patient to realize that he must have actually used his own muscular power. In these and similar ways the patient is ultimately convinced that his paralysis is disappearing, and as a result it actually does disappear.

The limitations and sphere of application of persuasion are much the same as in the case of suggestion. Like the latter, it is essentially dependent upon the production of the conviction aimed at, and is therefore also only suitable for cases where a conviction of converse type is the factor immediately responsible for the symptoms. It is superior to suggestion, however, in that it calls to its aid all the available forces in the patient's mind, instead of being merely thrust therein by extraneous powers. Like suggestion, it is extremely valuable when combined with other methods of treatment.

3. **Analysis** may be defined as the elucidation of the various mental causes responsible for a disorder, with a view to the removal of the

## PSYCHOTHERAPY

disorder by the removal or alteration of its causes. In this sense analysis has a very broad meaning, and must not be identified with psychoanalysis. The latter is, of course, one variety of analysis, but it is based upon certain definite causal theories devised by Freud, and the name should be limited to the particular mode of investigation and treatment initiated and elaborated by him. It should clearly be understood that all analytical methods owe a great debt to Freud, and that they are all based at least in part on the conceptions which he has introduced into modern psychology.

These various methods of analysis have as an essential common feature the fact that their main endeavour is to discover the precise mental causes responsible for a psychogenic disorder. They differ considerably in detail, but these differences are largely dependent upon the varying depths to which the investigation is pushed, some being content with the elucidation of the proximate causes, others insisting upon the further analysis of the proximate causes into their ultimate elements. The causal factors are frequently not on the surface of the mind, but buried as it were in its depths, and the problem is always to ascertain the nature of these deeper factors by the evidence accessible on the surface. The methods employed are therefore comparable to the procedures by which we deduce the nature of changes in the lungs from the signs, tactile, visual, and auditory, which are observed on the wall of the chest. For this purpose it is necessary to be acquainted with the various ways in which deeper mental factors influence the surface of the mind, so that the existence of the former may be deduced from the signs observable on the latter. In other words, a knowledge of psychological laws and mechanisms is required. In some cases the knowledge need merely be of a simple and everyday character, but in other cases analytical methods of treatment can only be carried out by the expert.

The sphere of application of analysis covers the whole field of the psychoneuroses, and possibly some part of the psychoses. It is indeed an essential constituent of any adequate psychotherapeutic treatment. In cases where mental conflicts play a predominant part in the causation, as for example in the anxiety neuroses, it is absolutely essential. In those conditions where suggestion and persuasion are important etiological factors—in hysteria and functional disturbances of various organs,

for instance—some degree of analysis is also necessary for completely satisfactory treatment, though much may be done here by the other methods already described.

*Psycho-analysis.*—Although the validity and utility of Freud's method of investigation and treatment are still the subject of great dispute, it is rapidly acquiring a dominating position in clinical psychology. It is not possible here to attempt any description of its nature and technique, nor indeed of any of the modern analytical methods. For this the reader must be referred to the literature.

4. **Re-education** is the process whereby a disordered function is trained or educated to return to its normal mode of activity. Properly speaking, it should not be regarded as standing upon the same level as the three processes already described, but rather as a secondary weapon used to complete the results achieved by the other processes. When, for example, an hysterical paraplegia has been removed by suggestion or persuasion, the patient will probably at first walk with an unsteady and defective gait. Re-education is then required to train the movements of the limb back to the normal standard of precision. Again, in a case of functional dyspepsia where the patient has become unable to take any but liquid food, the various mental factors which have produced the condition must first be dealt with, and then the digestive functions will require a gradual and careful re-education to accustom them once more to a normal diet.

**Combined methods.**—Although the four basic processes of psychotherapy have been described separately for the sake of clearness, they are in actual practice generally used in combination, and such a combination is really essential for efficient treatment. For this purpose the various mental factors which have produced the disorder have to be elucidated, and this process requires the employment of analysis. Certain cases only call for a comparatively superficial analysis, while in others the proximate causes thus elucidated have to be dissected into their ultimate elements. This is dependent upon the nature of the disorder and the individual character of the case, but in every instance the causes must be unearthed to the extent which will enable them to be dealt with. This investigation satisfactorily concluded, the next stage is the removal or modification of the causes thus discovered, and here suggestion, persuasion, and re-education are all capable of being employed. The physician

## PUERPERAL ECLAMPSIA

must, in fact, make use of every available agency to achieve the purpose of modifying the patient's mental processes so that they no longer lead to the production of disorder.

BERNARD HART.

**PTERYGIUM** (*see* CONJUNCTIVITIS).

**PTOMAIN POISONING** (*see* POISONOUS Foods, under POISONS AND POISONING).

**PTOSIS** (*see* EYELIDS, AFFECTIONS OF; VISCEROPTOSIS).

**PTOSIS, CONGENITAL** (*see* EYE, CONGENITAL ABNORMALITIES OF).

**PTYALISM** (*see* SALIVA, ANOMALIES OF SECRETION OF).

**PUERPERAL ECLAMPSIA.**—A state of convulsions occurring in a pregnant woman, and probably due to toxæmia of maternal hepatic or of foetal origin, or both.

**Etiology.**—The condition is invariably associated with albuminuria, which is really only one of the manifestations of a general toxæmia giving rise to a kidney lesion as well as to the convulsions. It occurs most commonly in first pregnancies, but may accompany a twin pregnancy in a multipara. When convulsions occur in a multipara they are usually the result of long-standing renal disease, and should be classed as uræmic rather than as eclamptic. The causation of eclampsia has been much debated, and an enormous amount of research of a chemical nature has been done on the subject. At the present time the disease is regarded as the result of a toxæmia either of maternal hepatic or of foetal, i.e. placental origin; it is more than likely that both influences may play a part in the same case. The underlying idea of the hepatic toxæmia of pregnancy concerns the function of the liver in relation to nitrogenous metabolism. If it can be assumed that one of the functions of the liver is to change nitrogenous waste material into some form in which it is easily excreted by the kidneys, then a failure on the part of the liver would result in the retention of these substances in the blood-stream, because the kidneys would be unable to excrete them. The result would be a general poisoning of the patient, an auto-intoxication, capable of damaging the kidneys, producing albuminuria, and by its action in the brain causing convulsions. It is not known what the poisonous substance may be, but it seems probable that it

is one of the antecedents of urea. Further, it is not known why the liver should fail in this manner, but it is possible that this organ is damaged by some poisonous substance of foetal origin, perhaps generated in the placenta. The placenta undoubtedly exercises excretory, as well as respiratory and nutritional functions, and consequently has an influence on nitrogenous metabolism comparable to that of the liver of the mother. If the placenta fails in this function, the maternal liver will have foetal waste products to deal with as well as those of the mother, and may break down or be actually damaged by the unusual strain thrown upon it. A further and somewhat curious way in which placental intoxication may be caused is believed to be by the introduction of foreign protein into the blood-stream, by the deportation of actual portions of chorionic villi. This fascinating observation—for deported villi have been seen in maternal organs—requires further confirmation before it can be finally accepted.

**Character of the fits.**—The convulsion is very like an epileptic fit, but differs from it in that there is neither aura nor cry. The fit consists of three stages, the tonic, the clonic and the comatose. In the first stage there is spasm of all the voluntary muscles, lasting a few seconds; in the second, all the voluntary muscles act with a jerky, twitching motion producing most irregular respiration and blueness of the patient from deficient blood aeration. The second stage, as a rule, lasts about a minute. In the third stage the patient is comatose, with stertorous breathing; its duration is about a quarter of an hour. In bad cases the patient may remain comatose between the fits for many hours. The number of fits is very variable, and, as a rule, depends upon the efficacy of the treatment adopted. During the fit the patient usually bites her tongue, and may injure herself by falling, striking her head, or burning herself on the fire.

**Premonitory symptoms** are usually well marked and of great importance, as their recognition enables treatment to be adopted which may prevent the occurrence of convulsions. Albuminuria and œdema of the face, limbs, vulva, and abdominal wall may precede the convulsions by a few hours or days. In some cases these symptoms appear weeks beforehand, but it must be agreed that in the chronic albuminuria and dropsy of pregnancy, eclampsia only follows in a small percentage of the cases. Headache, giddiness, disturbances of vision—weakness or partial blindness—vomiting, epi-

## PUERPERAL ECLAMPSIA

gastric pain and gradually increasing drowsiness are symptoms which usually herald the approach of the fits. In some cases the onset of the fit is so sudden that these symptoms may not have had time to appear or may have been unnoticed. Of more scientific value is the gradual diminution of the amount of the urea excretion and a gradual rise in the blood-pressure. These obviously can only be investigated when patients are found complaining of premonitory symptoms. During the fits the blood-pressure is always high; the urinary secretion often ceases altogether, and consequently urea excretion is in abeyance.

**Diagnosis.**—Usually there is no doubt as to the nature of the fit, but it must be remembered that a pregnant woman may have fits due to other causes. Among them must be placed *hysteria*, *apoplexy*, *meningitis*, *cerebral syphilis* and *cerebral tumour*. The differential diagnosis of these conditions is outside the scope of this article, but the main points in the diagnosis of eclampsia may be noted; they are the pregnant state, albuminuria, deficient urea excretion, considerable diminution in the urine, and great rise of blood-pressure.

**Prognosis** depends to a large extent on the treatment adopted, rapid delivery being always greatly in the patient's favour. In general terms, it may be said that many fits, continuous coma, high temperature, and delayed delivery are of grave import; whereas rapid and safe delivery with few fits, and a patient who is regaining consciousness and beginning to secrete urine, means almost certain recovery. The mortality is gradually diminishing with increased knowledge of the best treatment, and nowadays should be well under 10 per cent. It must be admitted that there are some cases in which the liver is so irretrievably damaged as to be incompatible with life, but unfortunately there is no way by which such cases can be recognized.

**Pathology.**—Only in the kidneys and liver are constant lesions found. Post mortem the kidney shows changes indicative of an acute tubal nephritis—cloudy swelling, necrosis, and loss of staining power of the nuclei of the lining epithelium of the convoluted tubules; the glomeruli are not affected. The liver shows areas of hæmorrhage and necrosis, sometimes microscopic but often large and conspicuous, secondary to thrombosis of the small perilobular veins and capillaries.

**Modes of death.**—Death is usually the result of exhaustion consequent upon the fits

and toxæmia. It may, however, occur from asphyxia, due to the excessive salivation and bronchial secretion which sometimes occur. This is particularly likely to happen if pilocarpine is used as one of the means of treatment: this drug should therefore be avoided.

**Treatment.**—If the case is seen in the premonitory stage, induction of premature labour by the bougie method or the use of a small hydrostatic bag is called for, the chief indications being increased drowsiness, scanty urine, and rapidly diminishing urea excretion. While waiting for delivery, give a saline purge, direct that large quantities of fluid be swallowed, and inject morphia hypodermically. Some authorities have advised the administration of thyroid extract in large doses, and claim good results from it. There can be no harm in giving a 5-gr. dose, watching its effect, and repeating it if it appears to be beneficial. Although it is important to deliver as quickly as possible, delivery must be accomplished with every precaution against sepsis and injury; the so-called *accouchement forcé* plays no part in the treatment of eclampsia.

When fits have occurred, treatment must be directed towards stopping or lessening their number, eliminating toxins, and re-establishing the renal secretion. Emptying the uterus stops the fits in the great majority of cases at once, so that this must be the first consideration. As **Cæsarean section** empties the uterus in a few minutes with perfect safety in the hands of a skilled operator, it would at first sight appear to be the ideal method of treatment, but experience fails to show that it gives better results than the less heroic plan to be described. Further, it is not always possible to perform the operation with safety, owing to the surroundings of the patient, the lack of a skilled operator or skilled assistance, or the absence of paraphernalia for aseptic technique, etc. Nevertheless, the operation has its place, and should be performed if the case is clearly desperate—one in which the fits have already been numerous, with continuous coma, and the patient is not in labour, but has a long, hard, undilated cervix.

To empty the uterus by **inducing labour**, time is required, during which the patient must have no fits, or as few as possible, and her blood-pressure must be reduced. Various drugs have been used for this purpose, e.g. chloroform, morphia, chloral and bromides, and veratrum viride. Chloroform is now ruled out

## PUERPERAL ECLAMPSIA

owing to its own dangerous effect upon the liver when administration is prolonged. Morphia has given moderately good results in the past,  $\frac{1}{2}$  gr. being injected hypodermically and followed by  $\frac{1}{4}$  gr. in two hours' time if the first dose has not had a pronounced effect. Chloral and bromide are practically useless, and may be left out of consideration. On the other hand, *veratrum viride*, of all the drugs used, has given the best and most constant results. In the form of veratrone, an aseptic solution containing the active principles in stable form, and given intramuscularly in a 1 c.c. dose, the drug produces most remarkable effects, lowering the blood-pressure, lessening the number of the heart-beats per minute, and usually stopping the fits for many hours. Though an extremely dangerous drug in a healthy subject, in eclampsia it seems to have no dangers whatever. As long as the blood-pressure is high, the patient plethoric, and the heart acting too quickly, veratrone may be given in the full dose of 1 c.c. To an anæmic patient with a blood-pressure only slightly increased (a very rare combination in eclampsia), only half the dose should be given. *Veratrum viride* has been used constantly in America in the treatment of eclampsia, but has never been much employed in this country owing to the unstable nature of the preparations and its consequent uselessness. Veratrone is stable, effective, and keeps for long periods.

The dose of veratrone having been given, the next procedure is to induce labour. This is best accomplished by rupturing the membranes and inserting a small hydrostatic bag into the amniotic cavity, first dilating the cervix with metal dilators, if necessary. It is noteworthy that the escape of the liquor amnii alone will often stop the fits, and is very good treatment. While one is waiting for the uterus to empty itself, the skin, bowels, and kidneys must be encouraged to act, the skin by a hot wet pack, the bowels by a large rectal saline washout; and the kidneys by hot applications to the loins, by making the patient drink if conscious, or by giving rectal saline infusion if unconscious. During this time she should be kept as quiet as possible in a darkened room, and no food should be given, only water being allowed.

These are the steps that have to be taken if a case of eclampsia is to be treated successfully, and success can only be looked for by carrying out the method in its entirety. As a rule, the uterus must be left alone to

## PUERPERAL FEVER

empty itself, and this can only be done with perfect safety if the patient is having no fits, the pulse has lessened in frequency, and the blood-pressure has diminished under the influence of the veratrone. If it is necessary to hasten delivery, forceps may be used when the cervix is fully dilated, or bipolar version may be performed when the os uteri admits two fingers easily, *but no damage must be done to the patient.*

Usually the symptoms clear up very quickly after delivery. The kidneys rapidly begin to secrete urine, and the albuminuria disappears within two or three weeks, leaving no permanent injury to the kidneys. Sometimes the patient remains unconscious for many hours or days, but as long as the kidneys are acting this need not be regarded as a serious symptom. Eye disturbances, even total blindness, may persist for some time, but generally these troubles disappear completely. They are due, as a rule, to a temporary albuminuric retinitis.

THOS. G. STEVENS.

**PUERPERAL FEVER.**—The term "puerperal fever" is a popular one for any disease which results from infection of the generative tract after labour. It includes such conditions as *puerperal sapræmia*, *septicæmia*, and *pyæmia*, with the local lesions which accompany them. Septic infection after abortion may also be included under the same heading, as the lesions and manifestations are similar. The minor degrees of infection are common, but the more serious ones show a slowly but gradually diminishing incidence as our knowledge of the means of preventing them increases. The use first of antiseptics and then of sterilized rubber gloves has led to a considerable diminution in the number of cases, but it must be admitted that this improvement has been more pronounced in lying-in institutions than in the private practice of doctors and midwives. For this the surroundings of the patients, especially of the poorer classes, are blamed, often quite unjustifiably. Open drains, cesspools, badly trapped water-closets, and dirty clothing have been looked upon as causes of puerperal sepsis without the least justification, and have nothing whatever to do with the actual exciting causes. These defects may conduce to chronic ill-health, and so render the patient less resistant to infection, but they are not concerned at all with the conveyance of infection. The actual source of the infecting agent lies in the patient herself, or in the hands, instruments, and appliances of the doctor or midwife. The

## PUERPERAL FEVER

patient herself is placed first because the infecting organism is not infrequently present in the lower part of the vagina and is probably derived from the intestinal tract. The commonest organism found in cases of puerperal sepsis is a streptococcus of the faecalis type, occurring in short chains, and found more often in the milder cases; while the *Streptococcus pyogenes*, in long chains, is more usual in the very serious types. There is no doubt that the infection in the vast majority of the cases is autogenous, in the sense that the organisms are present from the first in the patient's vagina or cervix; but it is also certain that digital or instrumental examinations and operations pick up these germs from the vagina and carry them up into the uterus. The fact that many cases of puerperal sepsis, both mild and severe, occur in patients who deliver themselves without any vaginal examination or operative interference is proof positive that autogenous infection must occur. In such cases it must be allowed that the organisms are at least present in the cervix from the beginning. The transference of infecting organisms from one patient to another must be a very rare occurrence now, although it was common enough in the past, when epidemics of puerperal septicaemia occurred in the practice of individual medical men. The proper use of sterilized gloves is an absolute safeguard against direct infection by the hands from one puerperal case to another, or from cases of erysipelas, septic wounds, septic throats, etc., to a lying-in woman. While the streptococcus is the usual organism and is practically always found, infections by other organisms, particularly the *Bacillus coli communis*, may occur. These, however, are secondary, both in incidence and in importance, to the ever-present streptococcus.

As in every other infective disease, the result depends upon the virulence of the organism and the resisting powers of the patient. When virulence is high and resisting powers are low, the worst forms of septicaemia result. Streptococci may be demonstrated in the passages of patients who show no symptoms of infection at all. The reason why lying-in women are prone to infection is found in the presence of the placental site with its open blood-vessels, and in the obvious or minute lacerations which in every case occur during delivery. Severe infection of a puerperal laceration, however, is a rare event, placental-site infections being much more frequent and serious. It is common enough to meet with a badly-infected perineal

laceration, but in such a case it is a practical certainty that the placental site is also infected, and is the real source of the important symptoms.

### PUERPERAL SAPRÆMIA

The minor cases of puerperal sepsis are usually classified as cases of puerperal sapræmia—the poisoning of the patient by the products of decomposition. They depend, as a rule, upon the presence in the uterus of a small fragment of placenta, membranes or bloodclot—dead tissues which have become infected with organisms and have undergone decomposition. They may consist merely of minute fragments of bloodclot adhering to vessels at the placental site, or may be obvious pieces of placenta. The infecting organism is the faecal streptococcus, of low virulence, which in the presence of dead tissue is a facultative saprophyte, and causes decomposition. The resulting products—chemical substances (ptomaines) and gases—are absorbed into the blood through the uterine wall and cause symptoms of septic intoxication. In certain cases shreds of devitalized tissue in lacerations may decompose for the same reasons, but this is not common. Although sapræmia occurs often enough in patients when no intra-uterine manipulation has occurred, manual removal of the placenta is a most important predisposing cause; it may be said with truth that this manipulation is almost invariably followed by some manifestation of sepsis and often by a very severe infection.

**Symptoms.**—On the second or third day after delivery there is a rise of temperature, slight at first but gradually increasing for two or three days. Usually the temperature does not rise higher than 101°–102° F., but when the decomposing material is large in amount very high temperatures may be met with. There is no rigor, and general symptoms, as a rule, are slight, perhaps a headache and some pain or tenderness over the uterus. The lochial discharge is free; it is usually offensive, and may be brown or almost black in colour. A definite sign is subinvolution, the uterus remaining at the same height above the pubes until the symptoms subside. There is no diminution in the amount of milk secreted. In the majority of cases, in which the decomposing material is small, the symptoms last from five to seven days; the temperature slowly subsides, the uterus gradually involutes, and complete recovery occurs, no local lesion of the uterus or adnexa persisting. In most cases no actual

## PUERPERAL FEVER

decomposing mass is extruded, the material being very small in amount and disappearing completely by solution during the process of decomposition. In a few, a piece of decomposing placenta, membrane or clot is passed and is seen on the pad; in a still smaller number of cases, perhaps in 5 per cent., the decomposing material has to be removed by a finger under an anæsthetic. Decomposition of a piece of chorion is a very rare event, and consequently if a piece of chorion is thought to be retained, it should be left alone; it nearly always slips out or breaks up and disappears.

**Results.**—The organisms causing decomposition rarely infect the endometrium, but if they should do so, involution is slow and a very mild chronic form of *endometritis* may result. This gives no symptoms at the time but may show itself by menorrhagia, leucorrhœa, and backache later, when menstruation is re-established. Slow involution and persistent red lochia for three or four weeks are common. *Severe sepsis* is very rarely seen when there is decomposing material in the uterus. In the worst cases the uterus is usually empty and the discharge inoffensive. Sepsis may, however, develop in a case which began as a simple sapræmia. That it occurs no more frequently, although the organism concerned is always a streptococcus, is proof of the high resisting powers of the patient in most cases. *Retroversion and retroflexion of the uterus* follows in many of these minor cases, owing to the soft subinvolved uterus falling back into the hollow of the sacrum and becoming permanently forced down by intestinal pressure on its anterior surface.

**Treatment.**—Active treatment for the interior of the uterus is very seldom required, as the symptoms subside spontaneously. All that is necessary is to sit the patient almost upright in bed so as to promote drainage from the vagina and prevent retroversion. Ergot should be given in the form of the liquid extract in doses of 1 dr. three times a day, with 10 min. of dilute sulphuric acid in water. Vaginal douches are uncalled for; they may not be harmful but are quite useless. Intra-uterine douches are also valueless, and may actually do harm by introducing more sepsis. They cannot dislodge adherent fragments of clot or placenta, nor can they disinfect the interior of the uterus. The only real difficulty is to recognize the cases which require digital exploration of the uterus. If the temperature is unusually high, say 103° or 104° F., and remains at this level for

two or three days, with very offensive lochia and a very tender uterus, the uterus ought to be explored. This must be done under an anæsthetic, the surgeon wearing sterilized gloves. The vagina must be prepared by swabbing it out with tincture of iodine, and the interior of the cervix should be treated in a like manner, first pulling it down with a volsella. The finger is then introduced into the uterus, the fundus being pushed down by the other hand on the abdomen, so that the finger may be enabled to explore every part of the cavity. Any retained tissue must be detached and hooked out with the finger; small fragments remaining may be broken up with the finger, but the curette should not be used. This instrument plays no part in puerperal sepsis, for the large flabby uterus opposes no resistance to it and tissues which the finger discovers and removes would be missed by the curette. Moreover, the softened walls of the uterus in the puerperal state are very easily perforated by it. After removal of any decomposing tissue, the uterus should at once be washed out with hot sterile salt solution (1 dr. to a pint), but this should not be repeated. There is no harm in using a solution of lysol or other non-poisonous antiseptic, but it does no more good than salt solution. Recovery is usually rapid after the evacuation of the uterus.

### PUERPERAL SEPTICÆMIA

In this group of cases the sepsis is more severe, the patient being poisoned by the growth and multiplication of organisms in the tissues and blood-stream, and by the toxins which they produce. The organism is always a streptococcus, either the long *Str. pyogenes*, or the short *Str. faecalis*. The point of entrance is usually the placental site, but in one form with a definite local lesion (pelvic cellulitis) it is through a laceration of the cervix. Although septicæmia may and does occur in unexamined cases and in unassisted labours, it is much more frequent in cases where there has been much intra-uterine manipulation, particularly in placenta prævia and in manual removal of the placenta. Manipulations inside the amnion are far less dangerous than those in which the fingers come into actual contact with the placental site.

Puerperal septicæmia affects the patient in exactly the same way as surgical septicæmia. Starting locally, the infection may spread by the usual channels, i.e. the lymphatics or the blood-stream or by direct continuity of tissues;



## PUERPERAL FEVER

it may be accompanied by important local lesions such as pelvic peritonitis and pelvic cellulitis, or it may prove fatal in a very short time by a generalized toxæmia without showing any evidence of a local lesion at all.

**Symptoms.**—The disease begins about the third day after delivery, practically always with a rigor, followed by a sharp rise of temperature and an increase in the pulse-rate out of proportion to the rise of temperature. The patient rapidly becomes very ill, may be delirious, perhaps has diarrhœa, has no appetite for food, and in fact contrasts very strongly with the patient suffering from *sapræmia*, who is never really ill. The worst cases end fatally within a week without the development of a local lesion; in them (and only in them) the milk secretion does not appear and the lochia become very scanty or may cease altogether. There are a very high temperature, a pulse-rate inordinately high, rapid wasting, repeated rigors, diarrhœa, vomiting, and delirium.

In the less severe cases, in which local lesions appear, the patient may be desperately ill with many of the foregoing symptoms, and yet treatment of local lesions as they appear often leads to complete recovery. The whole prognosis of puerperal septicæmia depends on the presence or absence of local lesions. These local lesions may be termed primary and secondary, all pelvic lesions being included in the former, while lesions at a distance, in the skin, joints, pleuræ, pericardium, meninges, etc., may be termed secondary.

**Primary lesions.**—The first local lesion is always a septic endometritis when the placental site is the point of entrance of the infection. From the endometrium the infection may spread by direct continuity of tissue to the uterine muscle (metritis) and to the cellular tissue of the broad ligament (pelvic cellulitis or parametritis). The latter, however, is more often the result of an infected laceration of the cervix, and in any case is now a comparatively rare disease. The second method of spread of infection is via the lymphatics, and as the lymphatics from the uterus communicate with the peritoneum, a peritonitis results; it is nearly always localized to the pelvis (pelvic peritonitis or perimetritis), but occasionally is generalized from the first. Pelvic peritonitis is by far the commonest local lesion in puerperal septicæmia, and is accompanied by local inflammations in the pelvic organs, particularly in the ovary and Fallopian tubes. Ovarian abscess is the most frequent

outstanding lesion found in operations for puerperal pelvic peritonitis; pyosalpinx occurs also, but is not so striking a feature. The ovary is infected directly through the lymphatics, but the tubal infection is from the peritoneum via the abdominal ostium.

When the infection spreads by the bloodstream its first effect is thrombosis of the uterine veins; this is followed either by thrombosis of the ovarian vein (commonly) or of the internal iliac vein. Extension of infection in this manner provides the examples of what is commonly called puerperal pyæmia or thrombotic septicæmia, which is rare, but quite distinctive. It is recognized by daily rigors and very wide excursions of the temperature, daily rises from normal to 104° or 105° F. being the rule. As the only local lesion is a thrombosed vein, which may suppurate, it is not at all easily detected. Although these local lesions are usually quite separate and distinguishable, cases are sometimes met with in which they coincide. Local peritonitis and ovarian abscess cannot develop without producing some degree of cellulitis, but cellulitis usually occurs without any involvement of the peritoneum at all. Thrombosed veins are always accompanied by more or less cellulitis of the perivascular tissues. In the mildest cases the only localized lesion may be a septic endometritis showing itself by subinvolution, pelvic pain and a purulent discharge from the uterus.

The **secondary lesions** may occur with lymphatic or thrombotic septicæmia, and differ in no way from those of surgical septicæmia. Arthritis, pleurisy, pericarditis, pneumonia, meningitis, abscess of the liver, and local abscesses in the skin may all complicate any case of severe puerperal septicæmia, and display their own distinguishing features. Among the minor lesions seen are suppurating wounds of the perineum and vagina, the so-called puerperal ulcers.

**Diagnosis and treatment.**—Diagnosis is not usually difficult, but occasionally it is not easy to distinguish septicæmia from some other intercurrent disease such as influenza, typhoid fever, pyorrhœa alveolaris, pyelonephritis, and other specific zymotic infections. The severity of the symptoms at the beginning, the rigors, and the extreme depression of the patient usually suffice to distinguish septicæmia from the milder cases of *sapræmia*.

In any severe case the first step must always be to find out the nature of the infecting organism, for purposes both of diagnosis and

## PUERPERAL FEVER

of treatment. With this object some discharge must be taken direct from the interior of the uterus and cultures made. It is sufficient to expose the cervix by a speculum, and then, holding up the anterior lip with a tenaculum, to pass a sterile swab on a wire into the uterus and withdraw it, soaked with discharge. It is practically impossible to avoid contamination with cervical organisms, in spite of the varied appliances invented by bacteriologists. The real organism, a streptococcus, will always be found, and if not pure can be separated by subculture. While doing this there is no harm in passing the gloved finger into the uterus to make sure that there is no gross retention of placenta, membrane, or clots, but it is useless to give an intra-uterine douche, and worse than useless to curette the uterus. If the uterus is making any effort to combat the infection, a protective barrier of leucocytes is marshalling in the endometrium; a curette will remove this important layer, will bare fresh areas to the infection, and produce a disastrous result.

A pure culture of the streptococcus concerned having been obtained, a vaccine must be made as quickly as possible. Vaccines will not cure the worst cases, nor will they prevent local lesions, but that they have lowered the mortality there is no doubt, and that the patient's general condition is improved by them is manifest. The vaccine must be autogenous, a stock vaccine seldom having any effect. In giving the vaccine it is usual to begin with a small dose of one million organisms, to repeat this in about thirty-six hours, to increase it to two million in about forty-eight hours, and then to be guided by the condition of the patient as to further increases and the duration of the intervals. As a rule, the larger doses are given at weekly intervals.

Nothing further can be done for the septicæmia itself, except to feed the patient as much as possible, to give stimulants when the heart shows signs of failure, and generally to aid the patient's resisting powers. It is not necessary to give drugs to bring down the temperature, and there are none which have any specific effects.

When the treatment of local lesions is in question much judgment is required as regards operative interference. General peritonitis should be treated by laparotomy and drainage as soon as diagnosed, but the results are very disappointing. Local peritonitis and ovarian abscess must be treated by laparotomy and removal of the ovary entire, if possible, along

## PUERPERAL INSANITIES

with the tube on the affected side, drainage being employed in all such cases. When there is no evidence of suppuration, many cases of local peritonitis clear up spontaneously, leaving numbers of pelvic adhesions. When an abscess forms behind the uterus and presses down Douglas's pouch, the safest treatment is to open the abscess through the posterior vaginal fornix. The fact that such abscesses are often ovarian does not alter the appropriateness of this avenue of approach. Pelvic cellulitis commonly undergoes resolution, but when it suppurates, the small abscess tends to point in the groin, following the round ligament. It should therefore be opened in that situation. When thrombosed veins have been the cause of the symptoms the question of an operation for their removal has arisen. Such operations have been recorded, and probably in the future will be done with greater success. It is a matter of experience, however, that many such cases recover without operations after long illnesses. The treatment of suppurative endometritis should be directed towards assisting drainage from the uterus, by sitting the patient up in bed. When the discharge is scanty the injection of hypertonic solution (5 per cent. sodium chloride with 1 per cent. sodium citrate, 2 oz. twice a day) will often be found useful in promoting the lymph flow and leading to resolution. To give the injection easily, a rubber drainage-tube may be passed up to the fundus and allowed to project at the vulva. This will drain the uterus and at the same time facilitate the injection of the solution.

THOS. G. STEVENS.

**PUERPERAL INSANITIES** (including those associated with pregnancy and lactation).—Under the term "puerperal insanities" are included different kinds of mental disturbance in which childbirth acts either as a direct cause, or as the final strain to an organism already predisposed to a mental breakdown.

Acute infective delirium has become comparatively rare as aseptic procedures during labour have been perfected, but milder confusional and exhaustive forms, mania, melancholia, and delusional cases occur. Either dementia præcox or paranoia may show its first symptoms during pregnancy, the puerperium, or lactation.

**Etiology.**—The most important *predisposing* cause is an insane or neurotic heredity; the unstable brain fails under the strain of emotion, physical exhaustion, hæmorrhage, or

## PUERPERAL INSANITIES

pain. *Exciting* causes are previous exhausting illnesses, worry, grief, the shame attending illegitimate birth and too frequent pregnancies. The insanity of lactation occurs chiefly among the poor, and may be induced by too prolonged nursing.

### **Symptomatology. Insanity of pregnancy.**

—Trivial eccentricities and deviations from the normal are common, varying from slight hysterical symptoms or exaggerated "longings" for peculiar kinds of food to definite delusions or melancholia. During the first few months there are occasionally depression and irritability, which usually pass off about the time of "quickening."

A more serious type occurs during the later months. Patients show symptoms of depression, fear, and anxiety, and become suspicious of their husbands, sometimes bringing charges of infidelity. This condition may develop into definite melancholia or delusional insanity, which continues after the birth of the child. In these serious cases asylum treatment is necessary, unless skilled and constant supervision can be carried out at home, not only on account of the danger of suicide, but because, with depressed and confused patients, the birth may take place without any noticeable pain and the child die for lack of attention. The danger of infanticide must also be considered.

During delivery there may be temporary excitement, and the patient may attempt to commit suicide or to kill the child, but after the birth the excitement usually subsides.

**Puerperal insanity** usually occurs within less than a week after labour, though it is sometimes delayed for some weeks. The infective type manifests itself about the third to the fifth day. The patient becomes restless, irritable, frightened, complains of strange thoughts or bad dreams, and sleeps very little. She is suspicious, jealous, often takes an aversion from her husband or child, and may attempt to kill the latter or to commit suicide. Hallucinations or delusions of poisoning may develop.

If the cause is septic infection, the mental condition is that of acute delirium, with rambling incoherent speech, restlessness, and visual hallucinations, accompanied by rapid pulse, high temperature, dry brown tongue, and constipation. There may be tenderness on abdominal palpation of the uterus, scanty and offensive lochia, and suppression of the milk.

In favourable cases the acute stage may not last more than a week, but it may be pro-

longed for a month or more. The excitement gradually subsides, sleep improves, and the patient begins to take an interest in her child and in what passes around her. In maniacal cases there is extreme excitement; the patient is noisy, sleepless, often has exalted delusions, and is difficult to control. This maniacal condition may last much longer than the delirious form, but complete recovery ensues in the great majority of cases.

In the melancholia that follows childbirth there is depression, with vague fears, anxiety, and frequently aversion from the husband and others. Delusions of persecution directed against herself or her child occur. These delusions of persecution may lead to violence and homicidal attacks. The depression may last for months or years, but recovery need not be despaired of if the general health is fairly good. Careful watching is, however, required for some time, on account of the danger of suicidal impulses occurring. In a few cases the condition becomes chronic and gradually sinks into dementia.

**Insanity of lactation.**—This usually takes the form of melancholia in exhausted, anæmic patients. The early symptoms are depression, fear, sleeplessness, jealousy, or aversion from the husband, sometimes followed by a tendency to suicide, infanticide, or homicidal impulses, or by delusions of persecution or sensory hallucinations.

In simple cases recovery usually takes place as the physical condition improves, but it may be delayed, or the patient may pass into a condition of chronic or recurrent insanity.

**Treatment.**—One of the first questions that arise is that of the desirability or otherwise of placing the patient in an asylum. If the mental disturbance occurs during pregnancy it is desirable to save the child from the stigma of being born in an asylum, and when the case is mild it can be adequately treated at home; all that is needed is care of the general health, cheerful surroundings, and as much freedom as possible from worry and anxiety. In melancholia the danger of suicidal tendencies, most difficult to guard against in a private house, must never be forgotten, and unless the patient is in a position to afford skilled nurses and is within easy reach of her medical attendant she can be far better treated in an institution.

At the first onset of mental symptoms during the puerperium the baby should be weaned and taken away.

If there be any symptoms of septic infection,

## PUERPERIUM, COURSE AND MANAGEMENT OF

every effort must be made to combat it at once. Should offensive lochia continue, or there be any possibility that fragments of placenta or membranes have been retained, the uterus must be explored and antiseptic douches given.

Everything possible must be done to conserve the patient's strength and to give her sleep. Fresh air and plenty of nourishing and easily digested food are essential, and, if she refuse the latter, tube-feeding must be resorted to unhesitatingly. Alcoholic stimulants such as brandy or port wine are occasionally needed in exhausted cases. Iron, arsenic, quinine, and strychnine are valuable as tonics, and cod-liver oil is often well tolerated.

Sleeplessness is a serious symptom. If it is possible to move the patient's bed into the open air during the day she will be more likely to sleep well at night, but in acute cases sedative drugs are needed. Paraldehyde is safe and most valuable, as it can be given repeatedly without ill effects. If there is no contraindication, such as hæmorrhage, a warm bath for 20-30 minutes at bedtime, at a temperature of 98° F., followed by a cup of warm milk, often has a calming effect.

In every case when mental symptoms appear, a complete physical examination should be made in order that no signs of disease be overlooked. Excitement or delusions may prevent a patient from complaining of pain, and mask the onset of some acute disease such as pneumonia. An examination of the urine may shed some light on the case.

E. M. JOHNSTONE.

**PUERPERIUM, COURSE AND MANAGEMENT OF.**—The puerperium may be defined as the period following labour during which the woman is recovering from the effects of pregnancy and labour and the pelvic organs are returning to their former condition. It occupies from six to eight weeks in normal cases, but may be prolonged in those which are complicated.

### I. THE MOTHER

**Physiology of the puerperium.**—The most important events in the puerperium are the involution of the uterus, the changes in the vagina and other pelvic structures, and the process of lactation.

**Involution of the uterus.**—After labour is completed the uterus can be felt by abdominal examination rising nearly up to the umbilicus or to about 5 in. above the top of the sym-

physis pubis. Intermittent contractions continue, but the organ remains firm and retracted.

During the first twenty-four hours or so little change is observed, but after this interval rapid diminution in size takes place; the fundus descends by about half an inch each day, though by a gradually diminishing amount, and by the tenth day can only just be felt above the symphysis. Afterwards the uterus becomes again a pelvic organ.

During the first week the cervix remains soft and the cervical canal easily admits two fingers; after a week it admits one finger only, and thereafter diminishes in size daily.

This process of involution is accompanied by changes in the surrounding structures. The vagina, the pelvic floor and diaphragm and the uterine ligaments, which have been stretched or torn during pregnancy and labour, gradually shrink till they approximate to their former condition.

It is important to realize fully the weakness of the pelvic floor and diaphragm after labour in order to appreciate the danger of prolapse which may result from putting a strain on these structures too early by a too brief confinement to bed. This weakness is due not only to the great distension of the genital canal during the passage of the child but also to actual tearing of fibres of the fascial planes traversed by the canal. Unless, therefore, adequate time is allowed for the healing of these torn fibres before any strain is thrown upon them, protrusion of the bladder or rectum, with the formation of a cystocele or rectocele, is liable to occur. The enlarged heavy uterus, sometimes retroverted, with its relaxed ligamentary supports, adds another factor to the important predisposing causes of prolapse.

**Lochia.**—In normal cases the lochial discharge during the first three or four days is red, consisting mostly of blood and decidual remains; the colour changes to brown as the quantity of blood diminishes, and gradually becomes paler during the next few days till, by the tenth day, it is colourless and very small in amount. When the patient gets out of bed for the first time there is often a slight return of the lochial discharge with reddish coloration. The lochia have a faint and characteristic odour, but if they become at all offensive, infection and decomposition are indicated. They contain no bacteria till they reach the lower part of the vagina, where non-pathogenic organisms are always present.

**Lactation.**—The secretion of milk is generally

## PUERPERIUM, COURSE AND MANAGEMENT OF

established on the third or fourth day of the puerperium. During the first two days there is no marked alteration in the breasts, but a small amount of colostrum is secreted; in it are large cells called colostrum corpuscles which contain fat globules. On the third or fourth day the breasts become swollen, tense and full, and the secretion of milk develops somewhat quickly. It is usually accompanied by a feeling of tenderness in the breasts, extending down the arms, and sometimes by a certain amount of general discomfort; it is not, however, associated with a rise of temperature. For the first few days the secretion is apt to be profuse.

**Urine.**—The urine not uncommonly contains a trace of lactose and albumin in the early puerperium. Contamination by the lochial discharge must not be forgotten, and if accurate examination is required a catheter specimen must be obtained.

**General management of the puerperium.** 1. **Rest.**—About an hour after the termination of labour the patient's toilet is attended to. Soiled towels and linen should be removed, the vulva should be covered with a sterile pad, a binder applied, and a hot drink given; the patient should then be encouraged to sleep. During the first twenty-four hours she should be kept as quiet as possible and may lie on her back or on either side according to her comfort, a change from time to time being desirable. After two or three days, more pillows are allowed and more movement permitted, and towards the end of a week she may be well propped up for meals; after this the semi-recumbent position is advantageous, as it promotes drainage of the lochia and facilitates urination and defæcation.

**Length of stay in bed.**—The advantages of an adequate rest in bed far outweigh any possible disadvantages. Under this régime the return of the pelvic organs to their former condition proceeds most advantageously, and the risk of permanent weakness of the pelvic floor with prolapse of the uterus is most satisfactorily countered.

If a definite plan be followed, allowing a gradually increasing amount of freedom and movement, a rest of three weeks should not be irksome.

During the first week the intervals between meals and between the feedings of the infant should be spent as much as possible in rest and sleep; during the second week, sitting up in bed may be allowed, starting at mealtimes,

and gradually increasing in length so as to avoid fatigue; during the third week almost any movements on the bed are permissible, and in addition massage and modified physical exercises should be arranged. Towards the end of this week the patient is lifted on to a couch and then to a chair, and afterwards walking may be begun and the normal manner of life gradually resumed. A warning, however, should be given that excessive fatigue must be avoided for another month at least.

During the early puerperium a binder adds to the comfort of the patient and assists the action of the bladder and bowels. A simple soft twill or linen band at least 18 in. wide is required, and may be fastened by safety-pins or by straps and buckles. It should reach down well over the hips and should not be applied at all tightly.

2. **Asepsis.**—Although puerperal infection most often results from interference during labour, yet it is quite possible to introduce infection during the early puerperium through lack of cleanliness or by making a vaginal examination. Such an examination should therefore be avoided for the first ten days at least, unless urgently indicated.

Should some complication arise demanding a vaginal examination or investigation of the uterine cavity, this should be performed with the same preparation and precautions as for a gynaecological operation.

For an ordinary bimanual examination, all necessary information can be obtained per rectum.

Asepsis of the genital canal is maintained by keeping the vulva clean and preventing the accumulation of the lochial discharge. On the completion of labour the vulva is swabbed from before backwards with 1-in-2,000 biniodide solution, and a sterile pad composed of absorbent wool covered with gauze is applied to it and pinned to the binder in front and behind.

The swabbing is repeated, and a clean pad applied as often as it is soiled with the lochia and after each action of the bladder or bowels, the number of pads required daily depending chiefly upon the amount of the lochial discharge.

Under normal conditions, if a soiled vulval pad be examined the red or brown colour is seen to be deepest in the centre and paler towards the edges; if, on the other hand, there is any infection of the lochia the stain on the pad will be deeper at the periphery.

## PUERPERIUM, COURSE AND MANAGEMENT OF

Particular care should be paid to cleanliness when any tear of the perineum is present.

**3. Management of the breasts.**—If the breasts are full and pendulous, comfort may be obtained from the use of a breast-binder. It must be applied loosely; it at all tight, it is not only uncomfortable but tends to check the secretion. Directions for the *care of the nipples* will be found under INFANT FEEDING.

The supply of milk may be too copious at the onset; if so, a saline aperient may be given once or twice a day; effervescing sodium sulphate, one drachm in half a tumblerful of hot water sipped directly after waking in the morning, is very efficacious.

For pain at the onset of lactation, aspirin (10 gr.) is very useful, but should only be given once from fear of checking the secretion.

**Involution of the uterus.**—If the uterus remains firm and contracted for an hour after the end of labour, the possibility of post-partum hæmorrhage may reasonably be excluded, and it is safe to leave the patient in the charge of the nurse. In order to ascertain that involution is proceeding normally, the height of the fundus above the symphysis pubis should be measured at the same hour each morning immediately after the patient has passed water. The height should be noted daily on the temperature chart, the 100° F. line being used for the level of the top of the symphysis and each degree above this representing one inch above the symphysis; by this means an accurate record is kept, and any delay or arrest of involution is noted. This may occur if any portion of placenta or membranes has been left behind or if any infection of the uterus is present.

**Ergot.**—A drachm of the liquid extract of ergot should be given in a little water as soon as the placenta has been expelled. Apart from this, it is unnecessary to prescribe ergot after normal labour. It may, however, be ordered with advantage when there has been hæmorrhage before, during, or after labour; when the lochial discharge remains persistently red or excessive in amount, or recurs when the patient gets up; or when subinvolution of the uterus is present. The following is a suitable prescription:—

R̄ Ext. ergot. liq. ℥ss.  
Syr. zingib. ℥ss.  
Inf. aurant. ad ℥i.  
Half an ounce t.d.s.

**The lochia.**—Inquiry should be made each

day as to the nature of the lochia, and the last pad should be inspected.

**Douches.**—Owing to the risk of introducing sepsis, routine douching during the puerperium is best avoided. Indications for douching, however, arise when the lochia become decomposed and offensive from infection of some part of the generative tract, or if hæmorrhage occurs. A hot douche, starting at 112° F. and rising to 115° F., if given by a trained nurse with all precautions, will cleanse the vagina of septic discharges and will stimulate the uterus to expel any retained secretions, clots, etc. Dakin's solution and eusol brought to the required temperature by the addition of boiling water are the best antiseptics, but if neither of these can be obtained, lysol (20 min. to the pint) or tincture of iodine (1 oz. to the pint) should be used. The douches may be given two or three times daily.

Intra-uterine douches should not be given except as part of a special operation when exploration of the uterus has to be performed.

**After-pains.**—These are not common after first labours, but may be troublesome in multiparæ. They are more likely to be severe after previous overdistension of the uterus or when the uterus is not empty. If they are acute and persist it may be necessary to prescribe an antispasmodic. Antipyrin 10 gr., repeated in two hours if necessary, is usually effective. Morphia should not be given.

**Diet.**—It is important to include plenty of nourishing fluids in the diet. During the first twenty-four hours milk, tea, bovril, cocoa, and custard are indicated. On the second day an egg and a small piece of fish or chicken may be added. On the third day, after the bowels have been opened, the diet should be increased to the normal, except that, in order to avoid flatulence and discomfort, the food should not be heavy. Meat and potatoes should only be permitted once a day. Stewed fruits may be allowed in moderation, but fresh uncooked fruits and salads should be avoided during the first weeks, as they are liable to produce intestinal disorders in the infant.

**Temperature.**—It is quite common for the patient to feel chilly and even have a shivering fit soon after the completion of labour; this is not, however, accompanied by any marked rise of temperature. The temperature should be taken at 8 a.m. and 8 p.m., and four-hourly during the day. After an ordinary labour in which no vaginal examination has been made the temperature remains within about a degree

## PUERPERIUM, COURSE AND MANAGEMENT OF

of the normal line. If it rises above 100° F., infection must be suspected, and if the rise occurs on the third day of the puerperium and persists for twenty-four hours with an accompanying increase of pulse-rate, the cause is most certainly infection of the genital tract. A rise of temperature cannot be caused by engorgement of the breasts or constipation *per se*; that resulting from the reaction to labour or from nervous shock is very transitory and uncommon. Though there is no doubt that during the puerperium the temperature is not stable, yet in a normal case the fluctuations from the normal are very slight. When a rise of temperature above 100° F. occurs, a careful examination of the patient should be made and, if no extraneous cause can be found, an aperient should be given and the further progress of the case awaited.

**Pulse.**—The pulse-rate should be noted on each occasion that the temperature is taken. After labour it returns to normal, and should remain so. In some cases it is rather slower than before labour. An increasing pulse-rate accompanied by a rising temperature indicates the onset of acute infection.

**Micturition.**—Difficulty in micturition results from the position of the patient, from the alteration in the anatomical relations occurring after labour, and from stretching, bruising or injury to the urethra during labour. If the bladder is allowed to become distended the difficulty is increased. After six to eight hours from the end of labour the patient should be encouraged to pass water. If she is unable to do so while recumbent, as is not uncommon, she should be raised gently to a semi-recumbent or a sitting position. Further aids to micturition are encouragement by the nurse, the application of heat to the lower abdomen, bathing the vulva with hot lotion, etc. In the event of failure it may be necessary to pass a catheter, but this should be avoided if possible, owing to the great risk of infection of the urinary tract. If a catheter is passed, a dose of hexamine (10 gr. in water) should be given at the same time. In exceptional cases it may be advisable after the first few days to allow the patient out of bed to pass water.

**Incontinence of urine** may be explained by—

1. Stretching or bruising of the urethra during a difficult labour.
2. Retention with overflow.
3. Vesico-vaginal fistula.

In the first case the incontinence is slight and partial and rapidly improves; in the second,

a full bladder can be detected and relief afforded by a catheter; in the third, incontinence is absolute and the patient has no control whatever.

In each variety of incontinence, hexamine, 5 gr. in water, should be administered three times a day as a precaution against infection of the bladder. Vesico-vaginal fistulæ often heal spontaneously.

**Bowels.**—About thirty-six hours after labour a dose of castor oil should be given to get the bowels open. Subsequently the amount of aperient required depends on the patient and the diet. In most cases some laxative is required to ensure an action of the bowels at least once every other day. Liquid paraffin is the best, if it is efficacious and the patient does not object to it; half an ounce should be given twice a day. If this is not sufficient, compound liquorice powder is useful. In obstinate cases some combination of aloes, nux vomica and belladonna (pil. aloin. co.) is the most satisfactory purgative, but it may affect the milk sufficiently to purge the infant or make the flavour distasteful. To minimize this risk, aperients should be given immediately after feeding.

**Sleep.**—It is important for the mother to have six hours' unbroken sleep each night, and care should be taken therefore that she is not disturbed by the infant during these hours. If sleep is prevented by pain in the breasts at the onset of lactation or by after-pains, the appropriate treatment should be given (*see above*). In other cases sleep can generally be induced by simple nursing measures. If the digestion is suspected a large dose of sodium bicarbonate ( $\frac{1}{2}$ –1 dr. in a little water) is useful. Morphia is never required. If sleeplessness persists it may be the precursor of mental instability.

## II. THE INFANT

**Care of the eyes.**—As soon as the infant is born the eyelids are carefully wiped with pledgets of wool soaked in 1-in-5,000 biniodide solution; after this it is wrapped up in a cloth or towel so that movements of the hands to the eyes are prevented. If gonorrhœa is suspected, two or three drops of a  $\frac{1}{2}$ -per-cent. solution of silver nitrate must be instilled carefully into each eye and washed out afterwards with normal saline solution; this is the routine practice in hospital and among district patients.

**The cord.**—After the infant has been bathed the cord should be religatured with a boiled ligature made up of three strands of

## PULMONARY TUBERCULOSIS

coarse thread. The stump, dried and powdered with boric-acid powder and dressed with sterile linen, is then folded up over the abdomen, and the binder is applied. This dressing is carried out each day after the bath. The cord separates by a process of dry gangrene in about five to seven days; if any damp spot remains after separation, it is advisable to continue the powder and dressing. When this treatment is carried out carefully complications due to the cord are rare.

**Bath.**—A daily bath should be given at a temperature of 100° F., a pure superfatted soap being used. At the first bath it is important to wash the face and head first so as to prevent contamination of the eyes. The vernix caseosa can easily be removed after preliminary smearing of the body with oil or vaselin.

**Binder.**—The binder should be made of flannel and should be stitched.

**Feeding.**—This subject, including digestive disorders, is dealt with fully under INFANT FEEDING.

**Weight.**—This is the most reliable guide to the health and progress. After a preliminary loss of 4 oz. or more during the first three or four days the weight at birth should be regained at the end of a week or at the latest ten days. After this a normal child will gain about 6 oz. a week.

GORDON LUKER.

**PULMONARY ABSCESS** (*see* LUNG, ABSCESS OF).

**PULMONARY EMBOLISM** (*see* LUNG, EMBOLISM OF).

**PULMONARY INCOMPETENCE** (*see* VALVULAR DISEASE, CHRONIC).

**PULMONARY OSTEO-ARTHROPATHY** (*see* OSTEO-ARTHROPATHY, PULMONARY).

**PULMONARY STENOSIS** (*see* HEART, CONGENITAL DISEASE OF).

**PULMONARY TUBERCULOSIS.**—This term comprises the pathological conditions resulting from the presence of tubercle bacilli in the lungs, pleuræ and bronchial glands, and their clinical manifestations. The alternative names "phthisis" and "consumption" still linger in popular use.

### ETIOLOGY

The bacilli found in the lung lesions and sputum of cases of pulmonary tuberculosis are

almost exclusively of the human type. In a few cases human and bovine bacilli have both been found, while in very rare instances the avian type has been described. These results form a striking contrast to the proportions in surgical tuberculosis and in the common localizations in childhood, such as glandular tuberculosis, in which, up to the age of 5 years, from 30 to 80 per cent. of the cases yield bacilli of the bovine type.

1. **Modes of access of tubercle bacilli to the human body.** Of the five possible methods, viz. inhalation, ingestion by the alimentary tract, entry by the tonsils, inoculation through the skin, and direct hereditary transmission, it is tempting to suppose that inhalation plays the major part. The great preponderance of the human type of bacillus in the lung lesions and sputum lends support to this supposition. Experiments by Calmette and others have demonstrated, however, that tubercle bacilli can pass through the intestinal mucous membrane without producing a local lesion, and be conveyed to the mesenteric glands, and thence to the bronchial glands. It is wise, therefore, at present to compromise and admit that both modes of infection occur, withholding judgment as to which is the more common. Entry by the tonsil to the cervical lymphatic glands and thence to the mediastinal and bronchial glands must also be admitted as a possibility. The other two modes of infection are comparatively uncommon and do not need discussion here.

There is now good reason to accept the view that under the ordinary conditions of life in most countries, the tubercle bacillus is ubiquitous and that infection is all but universal by the time adult age is reached. The evidence on which this view is based is given in the article TUBERCULOSIS FROM THE STANDPOINT OF PREVENTIVE MEDICINE.

The tuberculous lesions in early life are in the main glandular, the bones, joints, and peritoneum becoming involved later, or generalized tuberculosis following as a terminal event. On the other hand, pulmonary tuberculosis is rare in infants and young children, and when it occurs it is usually due to a direct extension from the bronchial glands. Pulmonary tuberculosis from the time of puberty onwards becomes the common and characteristic type of tuberculous disease. The explanation which is now gaining acceptance is that pulmonary tuberculosis is practically never due to a primary infection with the tubercle bacillus, but



## PULMONARY TUBERCULOSIS

that it is the expression of a reinfection either from without or from pre-existing lesions in the body. On this view practically everyone is infected in early childhood, either by milk from tuberculous cows or by inhalation of bacilli from human sources. Those in whom the protective mechanisms fail, die or develop the various manifestations of the disease so well recognized in childhood. The majority of the remainder acquire a protection sufficient to last throughout life under favourable conditions, while a minority develop pulmonary lesions later. On this view pulmonary tuberculosis is to be regarded as the expression of an incomplete immunity.

The question of infection and reinfection is of great importance, since it must influence profoundly the measures taken to prevent the spread of the disease. That the risks of infection from person to person in adult life are not great would seem to be shown by the oft-quoted statistics of the absence of any increased incidence in those working in hospitals and sanatoria for consumptives, and by those of infection between husband and wife. In regard to the latter, the general result of statistical investigation seems to be that conjugal infection is slight and to be accounted for where it occurs by "assortative mating" of those with predisposition to the disease.

While it is certain that the great mass of primary infection occurs in early life, it is unsafe to assume that there is no risk of adult reinfection.

**2. Influence of inheritance and predisposition.**—Since infection appears to be almost universal, while actual disease occurs only in a certain proportion of the population, it follows that the great majority of those infected must possess some protective mechanism, or conversely that those who succumb to the disease must have some special susceptibility or predisposition. Such conditions seem also best fitted to explain the variations in the character of the disease in different individuals. This condition of predisposition was postulated long before the discovery of the tubercle bacillus.

After Koch's discovery, the factor of infection was regarded as paramount and the influence of diathesis was ignored or disputed. The latter is now once more considered to be of great importance, though its exact nature is as yet uncertain.

The question of **heredity** is much discussed. It has long been recognized that pulmonary tuberculosis may have a pronounced family

incidence. Since direct transmission from parent to offspring is a rarity, the question arises whether family liability is due to greater opportunities for massive infection in early life owing to other members of the family suffering from open tuberculosis, or to the transmission of some predisposition or tendency. The general consensus of opinion favours the latter explanation.

The influence of *race* upon the incidence of tuberculous disease would seem to involve the same problem. It is tempting to assume that the differences in racial incidence depend upon the degree to which a relative racial immunity has been acquired as a result of exposure of the race to infection for longer or shorter periods. It has been repeatedly demonstrated that when tuberculosis first attacks a community it is a very acute and fatal disease.

**Sex and age.**—It is said that from the ages of 5 to 15, girls die from the disease more than boys, but that after this age there are more male deaths than female. Although deaths from this disease occur at any age, 90 per cent. fall between the ages of 15 and 65.

The **influence of other diseases** upon susceptibility is difficult to establish accurately, but is undoubtedly of importance.

(a) *The catarrhal group.*—Though supposed to favour the onset of tuberculosis, it is probable that the catarrhal form of onset of this disease is largely responsible for the supposed association.

(b) *Other lung diseases.*—Pneumonia is very rarely followed by pulmonary tuberculosis, and, when this sequence appears, the original pneumonia was probably of tuberculous origin.

Pleurisy, particularly the form with serous effusion, is so commonly tuberculous that here again the subsequent disease of the lung is generally a later manifestation of the same primary cause.

Pneumonoconiosis tends in its terminal stages to become tuberculous.

(c) *Infective conditions*, notably influenza, enteric, whooping-cough, and measles, are supposed to favour the onset of tuberculosis. In regard to influenza this has recently been denied.

(d) In some *cachectic conditions*, such as alcoholism and diabetes, tuberculosis of the lungs may occur as a terminal infection.

(e) *Syphilis* favours the onset of pulmonary tuberculosis, but, on the other hand, in syphilitic subjects it tends to a chronic course and fibroid type.

## PULMONARY TUBERCULOSIS

(f) *Heart disease.*—It is generally admitted that the subjects of congenital pulmonary stenosis are very liable to suffer and die from pulmonary tuberculosis. On the other hand it has long been supposed that the subjects of acquired valvular disease, especially mitral stenosis, are less likely to suffer from it. This is only true to a very limited degree, since the two conditions not infrequently coexist.

(g) Other diseases in which some degree of antagonism to tuberculosis has been assumed are *emphysema*, *asthma*, and *gout*, but if it exists it is certainly only partial.

(h) *Insanity.*—A distinct predisposition to tuberculosis of the lungs occurs among the insane.

(i) *Pregnancy* and the *puerperium* act unfavourably. The disease may remain apparently quiet during pregnancy, but after childbirth there is a rapid spread of the lesions.

(j) *Trauma.*—The fact that direct injury to the chest, severe blows and falls, may be followed by active pulmonary tuberculosis is well established, and the explanation is doubtless that the injury leads to the breakdown of previously arrested disease. "Gassing" may also lead to activity in old lesions, and possibly pave the way to fresh infection.

**3. Environmental factors.**—These are of great and sometimes paramount importance. They influence the factors both of infection and of susceptibility. Defective hygienic conditions, bad housing, lack of sunlight, deficient ventilation, overcrowding, and uncleanness increase the opportunities for the spread of infection and also lead to impaired resistance.

Some occupations lead to increased morbidity and mortality from pulmonary tuberculosis, notably those carried on in dusty conditions or in bad surroundings, such as quartz-mining, knife-grinding, printing, and the traffic in intoxicating drinks.

Overwork, underfeeding, anxiety, and physical or mental stress may all act as determining causes.

**Geographical, climatic, and telluric factors.**—These are of less importance than those just described. The disease can flourish wherever it finds a susceptible population. It is said that defective subsoil drainage promotes its incidence. It is more common and more fatal in town dwellers than in those in rural districts, especially among males. W. Gordon maintains that exposure to strong prevalent rainy winds leads to a higher mortality from this disease.

### PATHOLOGY AND MORBID ANATOMY

The earliest manifestation of the disease is the tubercle. In miliary tuberculosis it may form the chief morbid object apparent, whereas in the chronic forms of the disease it may not be seen at all or only in the more recent areas of the disease. Birch-Hirschfeld found that the initial deposit was usually in the walls of a bronchus of the third to the fifth order. The initial lesions are generally peribronchial, the tubercles developing in and around a small bronchiole. The early reactive changes are bronchitic and peribronchitic. The swelling of the bronchial wall leads to obstruction of its lumen, with collapse of the corresponding alveoli, and eventually to broncho-pneumonic changes. The subsequent course of these initial lesions depends upon the secondary changes which occur.

**Secondary changes.** (a) **Caseation.**—The tubercle is a non-vascular structure, and so far from leading to new vessel-formation, as some morbid processes may do, it produces occlusion of existing vessels. As a result of the diminished blood and lymph supply, and possibly partly in consequence of some toxic substances produced by the bacilli, the tubercles and the tissues in which they are deposited may become converted into a homogeneous mass of cheesy appearance and consistence in which practically all trace of structure is lost.

This caseous material may undergo one of two further changes, softening or calcification. In softening the caseous material is converted into a puriform liquid, constituting tuberculous pus. In calcification some inspissation of the caseous material occurs, and, later, lime salts are deposited. This change constitutes one form of arrest of the tuberculous process.

(b) **Fibrosis.**—In other cases the tubercles lead to reactive processes in the adjacent connective tissues, with new growth of fibrous tissue, so that the tubercles are completely enclosed. This constitutes a second and more satisfactory mode of arrest. Fibrosis may occur from the first, or may develop after caseation has been in progress.

**Modes of spread within the lungs.**—From the initial peribronchial deposit with its lobular extension the disease process may spread— (1) By direct infiltration at the margins. (2) By peribronchial extension, chiefly along the lymphatics. (3) By the subpleural and interstitial lymphatics. (4) By inhalation. Sputum containing tubercle bacilli may be inhaled into healthy bronchi and by implantation start fresh

## PULMONARY TUBERCULOSIS

foci. These are seen to have a lobular arrangement. This form of spread is not uncommon after hæmoptysis. (5) By the blood-vessels. By breaking down of a caseous focus into a vein, bacilli may be widely distributed throughout the lungs or even reach the general circulation, producing generalized miliary tuberculosis.

**Reactive changes.**—The reactive changes induced by the tubercles in the lungs are bronchitic, pneumonic and interstitial. The bronchitic processes lead to collapse and subsequent consolidation. In other cases the process is more acute and leads directly to large areas of *pneumonic consolidation*, either lobular or lobar, in which caseation rapidly proceeds, the resulting condition being referred to as caseous pneumonia. When caseation occurs it is very liable to be followed by softening. The *interstitial* changes are more pronounced when the tubercles are deposited in the peribronchial connective tissue, but they may occur around any tuberculous focus. Productive changes in the connective-tissue cells lead to the formation of strands of fibrous tissue which are often dense and pigmented.

Certain of these changes, viz. caseous pneumonia and excavation, require more detailed consideration.

*Caseous pneumonia.*—In acute caseous tuberculosis large areas of lung may become consolidated very rapidly, and histologically the appearances are somewhat different from the tuberculous broncho-pneumonia commonly seen—which is due to agglomerations of tubercles inducing lobular consolidation. In caseous pneumonia the exudate is more definitely inflammatory and contains fibrin.

*Cavities.*—It is by the liquefaction of caseous areas and the expectoration of the débris so formed that excavation occurs.

Cavities differ in appearance according to their age and size. They may be the size of a pea or practically involve the whole of one or more lobes.

A recent cavity is irregular in outline, and has rough shaggy walls composed of caseous material. It may be traversed by strands called trabeculae, which consist of bronchi and vessels wholly or partly obliterated, or, in later stages, of strands of condensed lung tissue. In very acute cases the process of caseation and softening may go on unchecked, forming very large cavities, but in most instances some reactive changes occur in the less involved tissues and a red line of hyperæmia may be seen just outside the wall of the cavity. New

formation of fibrous tissue occurs, forming a fibrous outer wall. Gradually the inner lining becomes smooth, constituting a thin false membrane, at first exuding pus but subsequently drying up almost completely. In this manner a chronic dry cavity is produced.

*Pulmonary aneurysms.*—Small aneurysms varying in size from a pin's head to a cherry are not infrequently found in cavities, usually in those of the chronic type. They are the most frequent cause of hæmoptysis in this disease. Ulceration of a vessel is also described as a cause, but this is rare. The aneurysms are usually found projecting into the cavity and arise from periarteritis weakening the wall of the vessel, particularly at its unsupported part.

*Arrest.*—In the great majority of post-mortem examinations in town dwellers, old arrested lesions are found at or near one apex. These vary in extent from slight thickening or puckering of the apical pleura with a small pigmented area of denser consistency in the lung tissue, to marked thickening and adhesion of the pleura with a hard fibroid nodule, a caseous mass enclosed in a dense fibroid casing, or calcareous nodules in the lung enclosed in pigmented fibrous tissue.

We may now consider the **morbid anatomy of the clinically recognizable forms of the disease.**

1. *Acute miliary tuberculosis.*—This condition may be confined to the lungs, but is usually part of a generalized tuberculosis. A primary caseous focus may be found at the apex of the lung, in the tracheo-bronchial, or intrapulmonary glands, or in some extrathoracic situation. The lungs are riddled with miliary tubercles, and death usually ensues before any secondary changes occur. The lung tissue near the tubercles shows broncho-pneumonic changes. If the miliary outbreak occurs as a terminal event in chronic pulmonary tuberculosis, the miliary tubercles are most closely clustered in the neighbourhood of the old lesions.

2. *Caseous tuberculosis.*—In this form, which is invariably acute, the lesions are chiefly those of caseous pneumonia, and the distribution may be either lobular or lobar, the latter being much the less common. The pneumonic patches form yellowish areas widely distributed throughout both lungs. In the lobar type the whole of one lobe and parts of the others may be affected. The consolidated areas are firm, dry, airless, and sink in water. The pleura over them is inflamed. Softening occurs rapidly, to form small cavities which frequently coalesce into large

## PULMONARY TUBERCULOSIS

irregular excavations with rough walls. If a cavity form immediately under the pleura before adhesions develop, escape of air into the pleura with the formation of pneumothorax may result.

**3. Fibro-caseous tuberculosis.**—In this the commonest form the appearances differ according to whether the caseous progressive processes or the fibroid retrogressive changes predominate. Both may be present in varying degrees in different parts of the lungs, and cavities of different size, age and character are usually present. The oldest lesions may show extensive fibroid change. If excavation is present in this part of the lung it is usually a chronic dry cavity. In other parts caseous areas may be present, softening to form new cavities. The line of march of the lesions in this form was described by Sir J. K. Fowler. The common site of the first lesion is about 1–1½ in. below the apex of the lung, rather nearer the posterior and outer borders. At an early stage the lower lobe of the corresponding lung becomes involved, usually at a spot 1–1½ in. below its highest point. From this spot the lesions tend to spread along the direction of the interlobar septum. The apex of the opposite lung is next affected, generally in a corresponding situation to the primary lesion. After this the lower lobe becomes involved. The middle lobe of the right lung frequently escapes, or may show only a few isolated lesions. In fibro-caseous tuberculosis pleural adhesion generally occurs over the oldest lesions and in the interlobar fissures.

**4. Fibroid tuberculosis** is not a sharply defined condition. Strictly speaking, it comprises all the varieties in which fibrosis predominates, from arrested lesions, too localized to give rise to recognizable clinical manifestations, to those in which fibroid changes occur after caseation and excavation. In the latter case, contraction, with displacement of the mediastinum, and compensatory emphysema of the healthy lung or of the sound parts of the diseased one, may result. The lesions found vary from small areas of dense fibrous tissue to contraction of the whole of one lobe or even the whole lung, with areas of fibrous tissue enclosing dried-up cavities, calcareous areas, or even inspissated caseous material. In this form of the disease the pleura is almost invariably greatly thickened and usually adherent. A factor in its production is often the inhalation of dusty particles, and the lung may show pigmentation from this cause. Bronchiectasis may develop, particularly in the lower lobe.

### Tuberculous lesions found in other organs.

**Bronchial glands.**—When the lungs are the site of tuberculous disease, foci are invariably found in the tracheo-bronchial glands, which are generally enlarged and deeply pigmented. The lesions found may be miliary, caseous, calcareous or fibroid.

**Pleura.**—The pleura is almost invariably involved. In acute forms and in early stages a dry plastic exudate occurs, which usually goes on to adhesion. In old extensive cases this may be universal and lead to great thickening of the pleura. This process also involves the interlobar fissures.

Sometimes in cases of active pulmonary disease, miliary tubercles may spread widely over the pleural surface and lead to a large serous effusion.

The *larynx* is frequently involved (*see LARYNX, TUBERCULOSIS OF*). Less commonly, tubercles are deposited in the trachea in late stages of the disease and give rise to widespread ulceration which occasionally extends deeply. Rarely superficial tubercles going on to ulceration develop on the tonsils, pharyngeal wall and buccal mucous membrane.

**Stomach and intestines.**—Tuberculosis of the stomach is exceedingly rare, while tuberculous ulceration of the intestine is common, especially in the lower part of the ileum near the ileo-caecal valve. The ulcers are chiefly found in the Peyer's patches and solitary follicles. They are often transverse to the long axis of the bowel and have raised edges shelving to the base of the ulcer, which is rough and nodular. Tubercles can often be seen on the peritoneal aspect. Similar ulcers may be found in the caecum and colon, especially the ascending part. Perforation and peritonitis may occur as terminal events, though rarely, considering the frequency of ulceration.

Tuberculous deposits may be found in the bones, joints, uro-genital organs, suprarenal glands, and in the central nervous system, in fatal cases of pulmonary tuberculosis. Other post-mortem appearances include atrophy and wasting of muscles, amyloid and fatty changes in the liver, wasting and fatty degeneration of the heart-muscle.

### SYMPTOMATOLOGY

**Onset.**—Certain modes of onset may be recognized:

(a) *Insidious.*—The patient complains of malaise and gradual loss of weight, with or without some small rise of temperature. There

## PULMONARY TUBERCULOSIS

may be at first slight or doubtful physical signs.

(b) *Catarrhal*.—A prolonged catarrh of the upper air-passages, or a succession of febrile "colds," should arouse strong suspicion of pulmonary tuberculosis.

(c) *With hæmoptysis*.—If the hæmoptysis is of any quantity, it indicates not the real onset of the disease, but a breakdown of an arrested lesion, or an incident in insidious disease previously unrecognized.

(d) *Laryngeal*.—Laryngeal tuberculosis is regarded as invariably secondary to pulmonary disease, but the laryngeal lesions may occasionally be active and progressive, while the pulmonary may be slight or inappreciable at first.

(e) *With gastric symptoms*.—Gastric symptoms may predominate in the early stages of pulmonary tuberculosis. If progressive loss of weight occurs, the temperature should be recorded and the chest carefully examined.

(f) *Pleural*.—The earliest symptoms to attract notice may be pain due to pleurisy—and in all cases of pleurisy the possibility of tuberculosis has to be considered, especially if serous effusion develops.

(g) *Pneumonic*.—This may occur in the acute forms, and is liable to divert attention from the real cause until the persistence of the fever, failure to resolve, and signs of softening demand reconsideration of the original diagnosis.

(h) *After injury*.—Traumatic pulmonary tuberculosis may occur with hæmoptysis or pneumothorax directly after the injury, or more insidiously weeks or months afterwards. Cases occurring after "gassing" may be included under this heading.

(i) *After other diseases*, more particularly the specific infections, such as measles, influenza, and pertussis. Here again the sequence may be immediate or more remote.

We may now consider individual symptoms in detail.

**Cough**.—In general miliary tuberculosis with meningeal symptoms, cough may be hardly noticeable; on the other hand, in local miliary extension of old disease within the lungs it may be almost incessant. In the insane, cough may be practically absent, though fever and active signs demonstrate acute spreading disease. In caseous tuberculosis, cough with copious expectoration goes on throughout the day and often disturbs the patient at night. In fibro-caseous cases the cough varies with the local condition, being frequent and troublesome when the disease is active, but becoming less

when it is tending to arrest, though in arrested cases with cavity or with extensive fibrosis a certain amount of chronic cough persists.

Cough followed by sickness is most frequent in the morning and may be due to the more tenacious sputum which occurs at that time irritating the fauces and leading to reflex vomiting. It is more common in early left-sided apical cases. A distressing form of cough of husky character occurs in laryngeal cases. It is often painful and associated with the expectoration of copious, frothy sputum.

A dry cough of reflex origin occurs in cases which develop dry pleurisy. Tuberculosis of the bronchial glands may give rise, at any rate in children, to a loud, harsh, barking cough without expectoration.

**Expectoration**.—The expectoration should be submitted to laboratory examination, or at least examined for tubercle bacilli. A complete investigation is concerned with the physical characters, the chemical composition, the microscopical examination, including the cytological and bacteriological features, and in doubtful cases with the results of inoculation into guinea-pigs.

The *amount* of sputum is extraordinarily variable. There may be practically none throughout the whole course of the disease, or there may be as much as 20 oz. or more in twenty-four hours in cases where active softening is in progress or with large spreading cavities. Patients who develop bronchitic changes (often due to secondary or non-tuberculous infections) may also bring up large quantities.

The **physical characters** of the sputum are equally inconstant. There may be only a little clear or turbid mucoid fluid, resulting from bronchial irritation or secreted by the walls of a chronic cavity, or copious quantities of tenacious muco-pus. In the early stages the expectoration may be of mucoid character with small streaks or beads of muco-pus or with small irregular yellow areas, probably of caseous material. These form the most likely material in which to search for the presence of tubercle bacilli. Small streaks or shreds of blood may also be present. When active softening is in progress, the sputum is yellow, and muco-purulent, and may come up in pellets which do not coalesce in the sputum cup but flatten out into discrete rounded masses, constituting the so-called nummular sputum. It is surprising how rarely the sputum in tuberculous disease is offensive, when the destructive nature of the

## PULMONARY TUBERCULOSIS

morbid process is considered. In cases where this occurs the disease is usually of fibroid character and has given rise to marked bronchial dilatation. More rarely, actual gangrene may have supervened upon the tuberculous process.

**Calcareous particles** are occasionally coughed up with the sputum, almost invariably in chronic cases. These pulmonary calculi vary in size from a pin's head to a pea. They are rough and irregular in surface. Occasionally calcareous masses as large as a small cherry may be expectorated. In many cases these are derived from an old focus in one of the tracheo-bronchial glands and may be associated with severe symptoms.

**Chemical characters.**—Chemical investigation of the sputum shows it to consist of water, mucus, salts, and organic substances. Sometimes Charcot-Leyden crystals and crystals of fatty acid are found. The presence of *albumin* has been investigated by Ridge and Treadgold, who found that practically all cases of active pulmonary tuberculosis contain albumin in the sputum and that its presence in doubtful early cases helps to support the diagnosis, while in the absence of tubercle bacilli a negative test for albumin on three successive occasions is strong evidence against active tuberculosis. It may, however, be present in conditions of heart disease, bronchiectasis and other conditions associated with alveolar damage.

**Microscopical examination.**—This is concerned with the demonstration of tubercle bacilli, the varieties of cellular elements and the presence of elastic tissue. Examination for tubercle bacilli is done by the *Ziehl-Neelsen* method or one of its modifications (see BACTERIOLOGY and PATHOLOGY, (CLINICAL)). If no bacilli are found by this method, Uhlenhuth's antiformin or concentration method may be employed. Antiformin (a mixture of sodium hypochlorite, sodium hydroxide, and sodium carbonate dissolved in water) is added to the sputum so as to make a 20- or 25-per-cent. dilution of the antiformin. The mixture is shaken, sedimented by standing or centrifuging, and the sediment examined by the carbol-fuchsin method.

If bacilli are found in a direct film, no very definite idea of the character of the process can be formed from the number of bacilli present, but a progressive diminution in the number in a series of examinations is possibly of favourable significance.

In films made direct from the sputum, the

number and character of the *cells* present may be studied. A differential count of these is said to show a large excess of mononuclear cells, though whether these are lymphocytes or modified alveolar cells is uncertain.

**Elastic tissue** is present when destructive processes are in progress.

Cultures of sputum may be made to determine if any secondary catarrhal organisms are present.

**Dyspnoea.**—In the early stages and where the disease is localized, dyspnoea is chiefly apparent on effort and is probably largely due to the lessened movement of the diaphragm on the affected side. In acute miliary or caseous tuberculosis, and in cases associated with high fever, it may in part be due to the effect of the raised temperature. In the terminal stages a cardiac factor may be added. A dry pleurisy may cause painful dyspnoea. In cases complicated by pleural effusion a mechanical factor is added, though the dyspnoea is often not so urgent as might be expected, unless the effusion is very large. Sudden and extremely urgent dyspnoea may occur when spontaneous pneumothorax develops.

**Cyanosis.** Cyanosis is a late rather than an early indication, and is directly related to the extent of lung tissue involved. It is naturally more pronounced in patients with pre-existing emphysema and in those with any cardiac affection.

The so-called " hectic flush " seen in acute febrile forms of the disease is not a cyanosis, but a vaso-motor dilatation incidental to the toxæmic, febrile state.

**Pain.**—Some cases, even those of acute type, may go through their whole course without any local pain, but in most cases it is present in some degree. The commonest cause is dry pleurisy; and pain of more or less sudden onset and influenced by the respiratory rhythm should always lead to careful examination. It is important to remember that the pain of diaphragmatic pleurisy is referred to the upper abdominal quadrants. Pain of dull, dragging character, often influenced by the weather, occurs in fibroid, or arresting cases, probably due to the contraction of fibrous tissue.

In cases with much wasting there is often tenderness of the skin, especially over the most advanced site of the disease. Severe coughing may cause pain in the upper abdominal muscles or in the region of their attachments to the ribs.

Pneumothorax may cause intense pain and induce fatal collapse. Meningitis may be ushered

## PULMONARY TUBERCULOSIS

in by severe headache. Cold abscesses about a rib or the sternum may give rise to local pain, and the cause may not be recognized until swelling and redness become apparent. Laryngeal cases suffer considerably from local pain, which is increased by coughing and by taking food.

**Night-sweats.**—Night-sweats of drenching character are peculiarly suggestive of pulmonary tuberculosis, occurring in this disease more frequently than in any other. It is only in acute febrile cases, or in the active stages of chronic ones, that they are very pronounced.

**Loss of weight and emaciation.**—Loss of weight is often a very early symptom, and should arouse suspicion, particularly in young adults. In acute disease, and in the late stages of the chronic forms, great emaciation develops, often very rapidly, and the patient's appearance becomes highly characteristic, with drawn flushed face, long neck with prominent pectoral muscles and Adam's apple, and band-like sterno-mastoids.

**Fever.**—Rise of temperature constitutes a most important indication of early disease, or of activity in previously quiescent lesions, and in all cases in which the diagnosis is in doubt a record of the daily temperature should be taken for at least a week.

**Form of temperature record.**—For most purposes it is sufficient to record the temperature four times in the twenty-four hours: (a) on waking in the morning (normally, the mouth temperature at 7 or 8 A.M. is 97°–98° F., and the rectal temperature 97.2°–99°), (b) at 1 P.M. after the hour's rest following on the morning exercise, (c) at 6 P.M., (d) at 9 P.M. after the patient has retired to bed. In some cases it may be found that the maximum temperature, which is usually from 4 to 6 P.M., may occur later in the day, even at 8 or 9 P.M.

When treatment has been carried to the stage that the patient is on full exercise, the morning and night temperatures may be all that it is necessary to take.

**Methods of taking temperature.**—In most sanatoria the rectal temperatures are taken and the Centigrade scale is used. The rectal record affords a more accurate measure of the body temperature than that taken in the mouth or the axilla. On the other hand, many patients prefer to take their temperatures in the mouth, and, if proper precautions be observed, the mouth temperatures give sufficient data for most practical purposes.

The temperature varies with the type and stage of the disease, and may depend upon

whether the patient is resting or taking exercise. Intercurrent affections may also influence the temperature chart.

In *acute miliary tuberculosis*, fever is almost invariably present and may be of continuous or remittent character. The so-called *typus inversus*, in which the morning temperature is the highest, is not infrequent and is of grave prognostic significance. Fever records of these types in the course of a chronic case suggest acute miliary extension, or pneumonic spread.

In *acute caseous tuberculosis* the temperature may at first be of continuous type and simulate the pneumonic chart, as the physical signs copy those of lobar pneumonia, but when softening and excavation supervene the temperature becomes typically hectic or intermittent, the diurnal variation being often 4°–5° F.

In *chronic fibro-caseous tuberculosis* the temperature record is variable. There may be so little fever that it escapes recognition, unless a careful record is taken over a period of days, or there may be none when the patient is at rest in bed, though a febrile rise occurs if he is up. An afebrile patient may develop a sharp rise of temperature, lasting one or more days and slowly subsiding, after extra exercise or at the change of grade of labour, and this may be associated with subjective phenomena like those from an overdose of tuberculin. Such rises have been proved to be due to what is called "auto-inoculation," and are attributed to an overdose of tuberculin from the patient's own lesions. The temperature chart is a useful index of progress and a valuable guide to treatment in these cases. If any acute spread or any caseation occurs the chart shows the features corresponding, as described above.

In *fibroid tuberculosis* the course may be practically afebrile, apart from auto-inoculations or any active spread.

Mention should be made of the association of fever with hæmoptysis. A considerable hæmorrhage may occur with little or no febrile rise following it, though sometimes a hæmoptoic bronchitis is set up by the inhalation of blood into healthy bronchi, which generally subsides in a few days. On the other hand, hæmoptysis may be followed by high and persistent fever. The hæmorrhage and the fever may both be expressions of fresh activity in old lesions, or the inhalation of blood containing tubercle bacilli may lead to a widespread outbreak of fresh tuberculous lesions in other parts of the lung.

The influence of menstruation upon the

## PULMONARY TUBERCULOSIS

temperature chart of women with tuberculosis of the lungs should be remembered. It has been found that a rise of temperature lasting from one to ten days may occur before the onset of the period. This premenstrual rise may occur to a less extent in apparently healthy women.

**Hæmoptysis.**—This is a symptom of great diagnostic value. Even tinging of the sputum should excite suspicion. Hæmorrhage occurs in about 50 per cent. of all cases of pulmonary tuberculosis. In the early stages it is usually slight, a few streaks occurring in the mucoid or muco-purulent sputum. In the pneumonic forms of caseous tuberculosis actual rusty sputum may occur. Profuse hæmoptysis is more frequent in chronic types of the disease than in acute, though it is sometimes present in the acute caseous forms. It may be so copious as to cause death from suffocation in a few minutes, but on the other hand, if it is not immediately fatal, a very large quantity of blood, amounting to as much as two or three pints in the course of a few days, may be lost and yet the patient recover. When the actual bleeding has ceased, the patient may bring up discoloured blood for some hours or days afterwards.

The exciting cause of hæmoptysis is usually stated to be some extra exertion, but Bang observed that in 69 per cent. of cases recorded by him it occurred while the patient was lying in bed or recumbent in a chair, while in only 6 per cent. did it occur during working or walking.

**Circulatory symptoms. The blood.** . . Although many observations have been made upon the blood in this disease, the results recorded have been so variable that no definite blood picture or sequence can be described. The erythrocytes may be diminished, but rarely to below 3,000,000 per c.mm. On the other hand, in cyanotic cases or in the course of treatment in sanatoria they may actually be increased.

A chlorotic type of anæmia appears to be the most common blood change. During treatment and in the later stages of the disease, the hæmoglobin percentage may be above normal; in the former case from super-alimentation, in the latter probably from loss of fluid and concentration of the blood.

The leucocytes may be slightly decreased in the early, and are increased in later stages, probably in part from the effects of secondary infections. It is said that leucocytosis is

usually present during softening, and invariably in cavity formation. When leucocytosis is present it is the polymorphonuclear forms which are increased and not the lymphocytes as in serous effusions due to the tubercle bacillus. Examination of the leucocytes by Arneeth's method (in which the polymorphonuclear leucocytes are grouped according to the number of lobes in the nuclei, from one to five) is said to give useful results. When the cells with fewer nuclei are increased, it is described as a deviation to the left. Taylor and Wilson have found that if there is much lævo-deviation in a case of tuberculosis, no great permanent benefit is to be expected from treatment, except prolongation of life; while if lævo-deviation is absent, the disease will probably be of chronic type. W. E. Cooke states that a left-handed deviation indicates toxæmia.

**Heart and blood-pressure.** In active stages and in the acute forms, the pulse-rate is often greatly increased, the patient as a rule being unconscious of this. The blood-pressure is commonly low in the active stages, sometimes 100 mm. Hg or less in bad cases. In cases progressing favourably it rises during treatment to normal, or supernormal. It should be remembered, however, that pulmonary tuberculosis may co-exist with conditions tending to raise the blood-pressure, such as arteriosclerosis.

The heart is usually small but there may be enlargement from right-sided hypertrophy. In the later stages symptoms of cardiac failure may occur. Edema of the ankles, when it supervenes, is a grave sign.

**Alimentary symptoms.** Pyorrhœa and various degrees of gingivitis are common in patients with tuberculosis seen in hospital practice, but there is no direct relationship. The association is, however, noteworthy, since bloodstained saliva, particularly in the morning, may be due to this cause and may mislead in diagnosis. Dysphagia occurs in cases with tuberculous laryngitis. The tongue may be clean and the appetite remain surprisingly good, even with a considerable degree of fever. Most patients, however, show dyspeptic symptoms at some time during their course. The usual forms are those which come under the heading of nervous or functional dyspepsia. They are generally due to defects in the gastric juice or to defective gastric motility. The chief symptoms are anorexia, discomfort and distension after food, flatulence, nausea and



## PULMONARY TUBERCULOSIS

vomiting. It is said that hyperchlorhydria occurs in the early stages, while the acid is diminished in the later period of the disease. The atonic condition of the muscular wall of the stomach may progress to actual dilatation.

In cases under sanatorium conditions, if prolonged hyper-alimentation is enforced, dyspeptic attacks develop, notably temporary intolerance of fats.

Intestinal symptoms are less common apart from actual ulceration, but constipation is frequent. Diarrhoea of troublesome character may occur, and does not necessarily indicate tuberculous ulceration.

**Nervous symptoms.**—The nervous condition of patients suffering from this disease is very variable. The *spes phthisica* is not common, and is practically confined to the very acute, caseous type. It is, however, striking when it is present. The patient is cheerful, optimistic, and expresses himself as feeling better, in spite of high temperature, cough, and progressive emaciation, and will speak of plans for the future when he is almost too weak to help himself. In chronic forms of the disease the patient is often emotional, sensitive, selfish and self-centred, although many patients devote themselves meticulously to the routine with its restrictions. Sometimes the mental changes progress to actual psychoses and alienation. The patient may become depressed or melancholic, delusions may develop, and suicidal impulses are occasionally manifested. Insomnia may supervene, but is usually the result of cough, dyspnoea, or night-sweats. Sometimes pronounced mental change, with headache, insomnia, or drowsiness, may usher in a terminal meningitis. Neurasthenia is a frequent concomitant of pulmonary tuberculosis, and may occur very early.

The peripheral nervous system is sometimes affected. The tenderness in advanced cases has already been mentioned. The reflex phenomena about the shoulder-joint described in detail by Pottenger are referred to under Physical Signs.

**Renal and reproductive systems.**—Apart from tuberculous deposits, few symptoms are observed in ordinary cases.

**Urine.**—The urine is normal in the early stages, but in advanced cases often shows abnormalities. In febrile cases it presents the usual characters, and often deposits urates. Albumin, sometimes with casts, is not infrequently present, and occasionally sugar,

even in cases without previous glycosuria or diabetes. Ehrlich's diazo-reaction is sometimes present. Urochromogen and indican estimations are of little practical aid.

In early stages, and in arresting cases, sexual desire is often strong. In advanced stages it is lost.

**Menstruation** frequently ceases quite early in the disease, especially if anæmia and debility supervene. It sometimes persists throughout until advanced stages and, if it has ceased, returns when the patient is improving. Tuberculous women remain fertile until a late stage of the disease.

**Physical signs. (1) Of early disease. Inspection.**—The patient may be of good physique and healthy appearance, or may show some definite signs of ill-health.

The hair may be lustreless and lank, the skin smooth and shiny, though it is usually dry. A growth of fine hairs upon the back, especially in children or young people, is suggestive and should arouse suspicion. Small dilated venules over the lower cervical and upper dorsal regions are frequently present and have been designated the "varicose zone of alarm," but no diagnostic significance can be assigned to them. If the points of the shoulders and the subcostal angle with the costal margins are separately and carefully watched during deep inspiration, some slight lagging in movement on one side or earlier cessation may give a valuable clue. Drooping of one shoulder, or hollowing or flattening above or below one clavicle, may serve to confirm this observation, as also may a hollowing of the supraspinatus region and wasting of the corresponding half of the trapezius muscle. The absence of lateral spinal curvature or kypho-scoliosis should be established before such observations are regarded as indicating early apical involvement. These conditions have been called the "shoulder phenomenon," and are attributed by Pottenger to a reflex effect comparable to the fixation of a tuberculous joint and the tonicities of the muscles around it.

Sometimes in women the breast on the affected side may appear slightly smaller than that on the healthy side. The position of the cardiac impulse should always be carefully noted if it is visible.

**Palpation.**—Vocal fremitus must be most carefully examined. Judgment is necessary in appraising the results obtained, particularly when the right apex is suspected, owing to the normal slight increase of vocal fremitus there.

## PULMONARY TUBERCULOSIS

If it is greatly increased on either side, a valuable piece of evidence has been obtained.

**Percussion.**—This should be light, since slighter differences in note are recognizable, and also because light percussion gives more information of the sense of resistance. Direct percussion over the clavicles is often useful. Percussion should be carefully carried out in detail, since even slight variations may be noteworthy in the presence of suggestive symptoms. Differences are particularly valuable in regard to the apical dome of lung extending above the clavicle. Percussion of this area and of the adjacent regions back and front, gives an area which is known as "Krönig's isthmus." Careful percussion may show that this area is narrower on the affected side by  $\frac{1}{2}$ –1 in. or more, and that the note over this area is slightly dull in early apical lesions. This is now widely recognized as a valuable early sign. It may be more easy to recognize slight differences in percussion note behind than in front, and the supraspinous and interscapular regions should be carefully compared. Some physicians pay very special attention to these regions as yielding valuable evidence of "hilus tuberculosis" (p. 48).

**Mensuration** is now little practised, but careful measurements or tracing of the contour of the chest by the cyrtometer may show some falling-in of the affected side.

**Auscultation.**—Breath-sounds that may be heard on the affected side are weak inspiration with expiration normal or inaudible, and cogwheel, wavy or jerky inspiration with expiration normal, inaudible or prolonged. Care must be taken not to mistake true "cogwheel" breathing, which is often irregular, for the regular interruption produced in nervous patients synchronously with the heart-beats. Inspiration may be harsh, with normal or prolonged expiration. A form of altered breath-sounds known as "granular breathing" has been described. It consists in a faint irregularity of inspiration suggestive of distant râles, though no actual râle may be distinguishable.

Various approximations to bronchial breathing may be heard, inspiration being of blowing character with normal or harsh expiration, and to these indeterminate forms the name of broncho-vesicular breathing is applied. Lastly, definite bronchial breathing may be heard, but this usually indicates that the lesions have progressed to actual consolidation.

**Adventitious sounds.**—Occasional small rhonchi or a few fine crepitant râles may be heard in the first deep inspirations or after cough, but

in early cases they are frequently absent, and in any case, if heard definitely and persistently, they indicate that the lesion is of some extent. Care must be taken to avoid being misled by extrapulmonary crepitant sounds, by atelectatic râles, by friction in cases of pleurisy, and by the fine bubbling râles audible on both sides close up to the sternum in emphysema.

**Voice-sounds.**—The same care should be taken in the appraisal of the voice-sounds as in regard to vocal fremitus, owing to the normal slight increase on the right side. Definite increase of voice-sounds, bronchophony and pectoriloquy audible above the clavicle, in the first two spaces or in the supraspinous region, are highly suggestive, and the further these are heard from the trachea in front and the spine behind, the more valuable they are.

It is in this early stage that the greatest difficulty in diagnosis occurs, and while none of the signs here described are characteristic, a combination of several of them coexisting with symptoms indicating some pulmonary affection is highly suggestive.

(2) **Signs of consolidation.**—On inspection, similar appearances to those described in the preceding section may be observed, but as a rule they are more definite. The flattening below the clavicle is more obvious and the limitation of movement on the affected side more apparent.

**Palpation.**—The vocal fremitus is now notably increased and the difference on the two sides is very pronounced, unless the consolidation is bilateral.

**Percussion.**—The percussion note is definitely dull, and if the area of consolidation is of any size the sense of resistance is notably increased.

**Auscultation.**—In consolidation at the apices, and at the upper part of the lower lobes, the breath-sounds are typically bronchial in character; but in the caseous type of case with acute pneumonic onset, and in cases in which pneumonic extension occurs in the lower lobes, tubular breathing may be heard. Adventitious sounds may be entirely absent, but crepitations or crepitant râles of fine or medium character are usually audible, or may be elicited on coughing. These râles may indicate that active softening is in progress, especially when they are abundant and of a coarse "sticky" character. The voice sounds are bronchophonic, and whispering pectoriloquy is invariably audible.

(3) **Signs of excavation.** *Inspection.*—If the cavity is in the upper lobe there is, as a rule, flattening and hollowing both above and below

## PULMONARY TUBERCULOSIS

the clavicle; the shoulder droops and the muscles of the shoulder girdle are wasted.

**Palpation.**—The vocal fremitus is increased owing to the consolidated lung tissue around, unless the cavity is full, or unless there is marked pleural thickening over it.

**Percussion.**—The percussion note may be dull if the cavity is small or full; tracheal, "boxy" or even "cracked-pot" in character if it is large, superficial, and in communication with an open bronchus. To elicit the cracked-pot sign the patient should have his mouth open, and percussion should be made sharply and rather forcibly. Even apart from the cracked-pot sound, opening the mouth may alter the pitch of the note on percussion over a cavity—this being known as Wintrich's sign.

**Auscultation.**—The breath-sounds may be bronchial, broncho-cavernous, cavernous, or amphoric, according to the size of the cavity and the amount of its contents; and in a basic cavity, particularly if it is due to a saccular dilatation of a bronchus from fibrosis, the breath-sounds are weak or absent if the cavity is full. Adventitious sounds are usually audible over a cavity; though if the cavity is dry they may be absent. Their character gives some indication of the state of the cavity, especially when correlated with the symptoms. There may be small, medium, or large crepitant râles, or coarse gurgling râles. Sometimes in a large cavity, metallic tinkling and amphoric echo may be audible. In total excavation of a lobe or of the whole of one lung, the bell-sound or *bruit d'airain* may often be elicited by means of two coins.

**Voice-sounds.**—Loud bronchophony and marked pectoriloquy are the rule over cavities, and are in some degree relative to the size of the cavity. Auscultation of the cough may demonstrate "post-tussive suction"—a hissing sound immediately after the cough.

(4) **Signs of fibrosis.**—When fibrosis is in progress, evidence of contraction of the lung and of displacement of the heart and mediastinum may be looked for.

**Inspection** often reveals pronounced asymmetry of the chest, and the affected side may be flattened, retracted and almost immobile. Compensatory spinal curvature may be noticed, with one shoulder much depressed. The cardiac impulse may be drawn upwards, and to the right or left. In right-sided cases it can be displaced nearly over to the right axilla, whereas in left-sided cases it may be in the

posterior axillary line, or even at the back, beneath the angle of the scapula. Owing to retraction of the lung, cardiac pulsation is not infrequently visible up to the second space in front. Dilated veins may be apparent over the front of the chest from displacement of the mediastinum and traction on the deep veins. The intercostal spaces may appear somewhat drawn in.

**Palpation.**—Vocal fremitus is diminished if there is much thickening of the pleura, but it is increased if there is consolidated lung tissue with open bronchi. The position of the cardiac impulse should be carefully localized, since its position affords valuable information as to the degree of fibrosis.

**Percussion.**—Over the fibroid area the note is dull, unless a large cavity is present in it, when it may be boxy. The sense of resistance is greatly increased. Owing to compensatory emphysema the resonance or hyper-resonance from the opposite lung sometimes extends across the sternum and encroaches largely upon the affected side. The cardiac dullness is sometimes difficult to separate from that due to the fibroid lung, but the pulsation visible from the uncovering of the heart will generally render its extent clear.

**Auscultation.**—Over fibroid lung areas the breath-sounds are generally weak, though if cavitation is present they are those given by the cavity.

Adventitious sounds are often entirely absent, but frequently small and medium râles are heard, often of sticky, metallic character. The voice-sounds also may be diminished, unless cavitation is present, when bronchophony and whispering pectoriloquy are heard.

It is important to remember that fibrosis can lead to definite bronchiectasis, especially when it occurs in the lower lobes, and that the characteristically variable physical signs of that condition may be present. If the cavity is full there will be absent or weak vocal fremitus and breath-sounds, with but few adventitious sounds or none at all, while if it is empty there is bronchial, cavernous, or amphoric breathing, exaggerated voice-sounds, and sticky, leathery râles or gurgles.

**Mensuration.**—Careful tracings made by the aid of the cyrtometer give a graphic record of the deformity induced, and may be used to trace the progress of the contraction.

(5) **Hilus tuberculosis.**—Of recent years an attempt has been made to separate out a special type of the disease in which the primary

## PULMONARY TUBERCULOSIS

ocalization appears to be in the immediate neighbourhood of the roots of the lungs, and the spread occurs along the bronchi and vessels in a wide conical extension. Its existence as a separate type is based largely on X-ray evidence. Clive Riviere maintains that the physical signs of this so-called hilus tuberculosis, though often slight at first, are always bilateral. He describes contraction of Krönig's area on both sides. Special parasternal and paravertebral areas of dullness are mentioned as indicative of glandular involvement. Riviere states that the earliest auscultatory signs may be heard at almost any part of the chest. Granular breath-sounds are often audible, but crepitant râles or other adventitious sounds develop only at a late stage. Excavation when it occurs is said to be in the middle of the upper lobe, the axillary region, or the base rather than at the apex. The X-ray photographs show distinct evidence of disease in the bronchial glands and radiating extensions in all directions. It is probable that this form of deposit and extension is the usual condition in the relatively uncommon pulmonary tuberculosis of young children, but it is at present open to question whether it is necessary to regard it as a separate form in the adult.

### (6) Occasional and non-characteristic signs.

—*Myoidema* may be observed at any stage, and is often more pronounced on the more affected side. It is best obtained over the pectoral muscles. It is most prominent when emaciation is present, but may be noticed early in the disease.

*Clubbing of the fingers* occurs commonly in chronic cases. The usual form is longitudinal curving of the nail, giving it a parrot-hill appearance, but sometimes the "drumstick" form of clubbing occurs; this is usually in fibroid cases, and especially in those associated with bronchiectasis.

*Pulmonary osteo-arthritis.*—In chronic cases, especially those with basal bronchiectasis, extensive joint-changes are occasionally found in addition to pronounced clubbing. There is at first swelling about the wrists and ankles, and later about other joints, including the knees and elbows, rarely the hips and shoulders. Sometimes considerable effusion occurs into the affected joints. Much deformity and limitation of movement may result, but pain is usually slight or absent.

*Cardio-respiratory murmurs* are often heard over a wide area in this disease. Pleuro-pericardial friction is sometimes audible over

the region where the pericardium and the pleura are in contact.

(7) *Radiography.*—X-ray examination of the chest has become established as an additional method of physical examination. To get the maximum aid from this method the chest should be examined by the fluorescent screen, and also by the taking of radiograms. The results of both must be interpreted in the light of the symptoms and physical signs.

The screen gives information as to the respiratory movements of the diaphragm, the degree of brightening of the lungs during respiration and cough, and also as to the position, shape and movements of the heart. Unilateral limitation of movement of the diaphragm was described by Williams as a sign of early disease, but it may be observed in other conditions, such as pleural adhesion, and is therefore not characteristic.

The radiograms may show the extent and distribution of the lesions, and demonstrate the presence of caseous areas, cavities, pleural thickening or adhesion. They are also of great assistance in confirming the existence of complications such as pleural effusion and pneumothorax. In establishing the existence of early lesions they are of less value. Slight differences in the appearances at the apices may be due to old quiescent lesions, and definite clinical signs may be present in cases without appreciable X-ray evidence.

Great attention has been paid to the shadows at the roots of the lungs. It must be admitted that there is still some difficulty in interpreting these root shadows. It is often easy to recognize calcareous deposits in the bronchial glands, but considerable experience is necessary to decide whether there is peribronchial thickening, and to distinguish between the shadows of the bronchial walls and vessels, and those cast by abnormal developments of fibrous tissue.

The character and situation of the heart shadow should always be studied. The former is often narrow, vertical or "drop"-shaped, and it is usually small. Displacements of the heart are well shown.

Exaggerated claims for X-ray indications have been made by some enthusiasts. It should be remembered that the appearances observed are shadows, not pictures, that conditions of widely differing origin may give similar shadows, and that catarrhal processes and the earliest lesions do not obstruct the rays unless there is widespread miliary deposit. In the presence of symptoms and signs the X-rays

## PULMONARY TUBERCULOSIS

may be of great value; they often indicate the extent of the disease and reveal the site of lesions of which the ordinary signs may be inconclusive or obscure. With our present knowledge, however, they cannot be regarded as an infallible means of early diagnosis, nor do they enable us to distinguish certainly between quiet and active lesions. They should never, therefore, be the sole or even the predominant factor in diagnosing tuberculosis of the lung.

### COURSE

In *acute miliary tuberculosis* fever, wasting and exhaustion, or generalization of the infection, may bring about the end in from one to three weeks.

In the *acute caseous* type death usually occurs within six months, unless artificial pneumothorax treatment is possible.

In *chronic fibro-caseous tuberculosis* the course is variable. During treatment such cases may proceed to complete arrest, or there may be arrest for a time and then relapse, or the disease may slowly advance. The duration may extend into several years, with long periods of freedom from pyrexia and almost complete arrest, alternating with bouts of fever and symptoms varying in severity according to the activity of the lesions.

The *fibroid* type may be practically afebrile throughout and the duration extend into years, even when complete arrest does not take place.

These clinical types, however, are not sharply delimited, and may merge into one another.

Some of the attempts to classify the course of the disease into stages are described in the general article on Tuberculosis.

### COMPLICATIONS

1. The complications due to the spread of the bacillus may result from its direct implantation in other organs, as the larynx and intestine, from direct extension and by lymphatic spread, as in pleurisy, pneumothorax, and pericarditis. Others are due to bacilli gaining access to the blood-stream and causing disease at a distance.

Tuberculous laryngitis is described elsewhere (see LARYNX, TUBERCULOSIS OF). Much less commonly foci occur in the trachea, the pharynx, tonsils, fauces, and buccal mucous membrane, rarely in the nose and ear.

Bronchitis is a not uncommon complication, due to the tuberculous process itself or to secondary infections.

Bronchiectasis sometimes results from fibroid disease. Gangrene of the lung occurs but rarely.

Emphysema is found under two conditions: one as a compensatory process in the unaffected parts of the lung, the other as an antecedent of the tuberculous disease.

Dry pleurisy is very common, and is a frequent accompaniment of a fresh outbreak or extension of the disease. It may proceed to effusion, which is not necessarily unfavourable, since it may keep the affected lung at rest.

Empyema occasionally results, either from invasion of the pleura by some pyogenic organism, or possibly from the tuberculous process itself.

Pneumothorax, one of the most serious of all complications, is usually caused by rupture of a subpleural caseous focus in a part of the lung where there are no pleural adhesions, or where if they exist they are localized and recent. Pneumothorax may be partial or complete, and the opening in the lung may remain patent, become valvular, or close completely. The air may be absorbed, but more commonly pleurisy is induced and hydro- or pyo-pneumothorax results.

A cold abscess about a rib or costal cartilage may constitute a painful complication.

Pericarditis occurs, but is not common. Myocardial weakness is frequent towards the end of chronic cases, and at autopsies small recent vegetations are sometimes seen on the mitral or aortic valves.

Intestinal ulceration is common in chronic cases and may cause pain, diarrhoea, offensive stools, and eventually emaciation. Perforation of an ulcer occasionally occurs, but is unusual. In chronic cases a hypertrophic condition develops in the caecum and adjacent part of the ileum, leading to a mass palpable through the abdominal walls.

Ischio-rectal or perineal abscesses going on to produce fistula in ano are common. These conditions are most likely to be met with in the later stages, but they may be a very early manifestation. Ulceration of the rectum occurs occasionally.

Appendicitis is not frequent; it may be due to the tuberculous process or to some other infection. Peritonitis is rare in pulmonary cases apart from intestinal ulceration, perforation, and appendicitis.

Other complications are due to distant localizations of tubercles, as in the kidney, bladder, or genital organs. Of these, deposits in the epididymis are the most frequent.

## PULMONARY TUBERCULOSIS

Spinal tuberculous lesions sometimes develop, and give rise to pain, limitation of movement, and cold abscesses.

Addison's disease is a rare association.

Meningitis is the most serious of all complications, since it is almost invariably an expression of generalization of the bacilli.

Among not infrequent intercurrent infections are influenza, catarrhal conditions of the air-passages, and lobar pneumonia.

Asthma occurs in patients with tuberculous disease, and may be induced by it. Diabetes may be a complication, but is usually the primary condition, the tuberculosis being secondary. Amyloid disease and fatty liver are now less often found than formerly.

Cold blue extremities and troublesome chilblains are frequent in tuberculous patients. Erythema is an occasional late complication.

### TERMINATIONS

The various terminations may be described as—

1. *Complete and permanent arrest.*—The disease process may be completely encapsulated by fibrous tissue, or, if caseation has occurred, calcification and fibroid changes may take place and completely close the lesions.

2. *Partial or incomplete arrest.*—The only evidence that arrest is not complete may be slight cough and expectoration with tubercle bacilli present and small febrile reactions after exertion.

3. *Rapid progress of the disease.*—In the acute form the lesions may spread rapidly and the toxic symptoms dominate the picture.

4. *Death* results from the disease or from its complications in a large proportion of its victims, though at very variable times after its onset. The most common form of death is from progressive asthenia, emaciation, and now cardiac enfeeblement. Death by asphyxia is not common but may occur in miliary tuberculosis and in very severe hæmoptysis. Death from hæmoptysis itself is less frequent than might be supposed, occurring in about 2 per cent. of cases. Death may result from complications, usually the result of spread of the tuberculous process, e.g. tuberculous meningitis, perforation from tuberculous enteritis, asthenia and wasting from starvation through laryngeal involvement, and pneumothorax. Death may also result from intercurrent disease, such as ordinary pneumonia, influenza, or bronchitis.

### DIAGNOSIS

The combination of characteristic symptoms with definite physical signs and the presence of tubercle bacilli in the sputum constitute a chain of evidence which is complete and incontrovertible. Unfortunately the cases in which such a conclusive demonstration is possible are already past the earliest stage.

It would be difficult to make a complete list of the various conditions simulating some phase or other of this disease, but it is useful to consider first those in which the symptoms are suggestive and the physical signs are indefinite or obscure, and secondly those in which definite pulmonary physical signs are present like those of tuberculosis. Among the former group may be mentioned chlorosis, dyspepsia, enteroptosis and chronic intestinal stasis, neurasthenia, Graves's disease, conditions of the nose, pharynx, larynx, and bronchial glands. Among the latter group, the following may give rise to difficulty in diagnosis, viz. asthma, pneumonia, broncho-pneumonia, apical catarrh or collapse, emphysema, fibroid disease or chronic pneumonia, pneumoconiosis, abscess, gangrene, bronchiectasis, pleurisy with effusion, syphilis, actinomycosis, hydatid or other pulmonary parasites, intrathoracic malignant disease, lymphadenoma, and oedema or infarction of the lung.

In the first group the diagnosis can usually be established by careful search for suggestive physical signs of early disease, by observation of the temperature, and by repeated sputum examinations. Specific tuberculin tests may also be employed in special cases. Examination of the blood, the naso-pharynx, larynx, thyroid body, or digestive tract may reveal the cause of cough, malaise, wasting, rapid pulse, etc.

In the second group, in which definite physical signs simulating tuberculosis are found, special methods of diagnosis may be necessary. In general, it may be stated that when repeated sputum examinations for tubercle bacilli are invariably negative, the diagnosis should be reviewed.

In cases of *asthma* the variable physical signs and the nocturnal paroxysms may give the clue, but it should be remembered that asthma may coexist with tuberculosis, or even be induced by it.

In *pneumonia* the course of the disease and culture of the sputum may settle the diagnosis, but tuberculosis should be suspected in any case of delayed resolution.

## PULMONARY TUBERCULOSIS

*Apical catarrh* after influenza or in association with emphysema may require several examinations and repeated sputum tests to establish its nature. Unilateral diminution of Krönig's isthmus and flattening or diminished movement would be very suggestive of tuberculosis. *Fibroid conditions of the lung* secondary to pneumonia, pleurisy and other non-tuberculous conditions may give rise to great difficulty, particularly when they occur in the apical regions. The history and repeated sputum examinations afford the best aids to diagnosis.

Similarly in regard to *abscess, gangrene, and bronchiectasis*.

In *pleurisy with effusion*, bronchial breathing and râles are often heard in the collapsed lung over the effusion. The absence of tubercle bacilli from the sputum and the disappearance of the auscultatory signs with absorption of the effusion may establish the real nature of these signs.

*Syphilis* of the lung is rare, and should be diagnosed only in cases with a definite history of this disease, a positive Wassermann reaction, and signs of intrathoracic disease of chronic type with repeated negative sputum examinations.

*Actinomyces* gives rise to symptoms like tuberculosis and to similar signs, though more often in the lower and middle lobes. The diagnosis depends upon the discovery of the specific organism.

*Hydatid disease* of the lung may be revealed by X-ray examination, the cyst giving a characteristic rounded shadow, or by the discovery of hydatid hooklets in the sputum.

*Intrathoracic malignant disease* may give rise to great difficulty, but the development of pressure signs, the stony dullness, the absence of tubercle bacilli, and the rapid development of effusion in some cases may give useful indications. The X-rays may be of great value in this connexion.

*Lymphadenoma* of the intrathoracic glands is often associated with periodic febrile attacks, and may be difficult to recognize until other glands enlarge.

*Infarction* of the lung giving rise to hæmoptysis may at first suggest tuberculosis, but the condition causing it is usually apparent.

When, after a careful review of the history, the symptoms, the signs (including X-ray findings), the temperature range, and the characters of the sputum, the diagnosis remains doubtful, recourse may be had (1) to the tuberculin tests, (2) to blood or serological tests.

**1. Tuberculin reactions.**—These reactions are sufficiently described elsewhere (*see under SEROLOGICAL DIAGNOSIS*), but it must be pointed out that while their specificity is established, except in the case of the *subcutaneous* test they are too delicate, and indicate old infection without differentiating between activity and quiescence. With the subcutaneous reaction a positive result with the weaker dilutions gives definite suggestion of active disease, which is conclusive if a definite focal reaction is apparent. It cannot, however, be applied in cases with fever, and if injudiciously employed it can do harm by the intensity of the focal changes induced. *Pirquet's* test is so sensitive that it is only of real value in the first two or three years of life, when as Riviere happily expresses it, "infection is synonymous with disease." Attempts have been made to increase its value by a quantitative modification. *Calmette's* test is less sensitive, and is not constantly positive in cases which are clinically tuberculous.

A further fact detracting from the value of tuberculin tests in diagnosis is that they are negative in acute tuberculous infections and in advanced stages, also during some infectious diseases. In the writer's opinion they should seldom be employed in the diagnosis of the pulmonary form of tuberculosis.

**2. Blood and serum tests.**—The complement-fixation test (*see SEROLOGICAL DIAGNOSIS*) has been applied to the diagnosis of tuberculosis. At present it has not reached the stage of affording a reliable criterion of active disease.

The opsonic index to tuberculosis has also been employed, and in expert hands may possibly be of value. It necessitates an examination of the index before and after exercise, and is not convenient for general use.

### PROGNOSIS

In endeavouring to form a prognosis, the practitioner should review all the features of the case, including the family and personal history, the type of the disease, the symptoms, especially the temperature record, the physical signs and their extent, the sputum, the blood reactions if available, the occurrence of complications, and the response to treatment.

**Family history.**—A bad family history of this disease is generally regarded as unfavourable.

**Personal history.**—A history of *alcoholism* is ominous. The patient is not likely to take treatment seriously, his digestion is often gravely impaired and his nutrition poor.

## PULMONARY TUBERCULOSIS

A previous history of *syphilis* is not necessarily of serious prognosis, since in such patients tuberculosis tends to fibrosis.

*Diabetes* influences the prognosis gravely and, if of severe or composite type, renders treatment and diet difficult.

*Gout* is an uncommon association, and seems to be of favourable influence.

*Mitral stenosis* has been regarded as favourable, while *pulmonary stenosis* and other forms of congenital heart disease promote the onset of the disease and influence its course adversely. *Cardiac hypertrophy* is usually a good sign.

*Insanity* is a serious association, and often leads to a rapid febrile course.

**Age.**—In infants and young children, pulmonary tuberculosis is rare, but the prognosis is bad. After the age of 15, prognosis is serious, at any rate till the age of 20 or after. From this time till about 45 or 50, age seems to be of slight influence in prognosis, but after 50 the disease is liable to be progressive.

**Sex.**—Sex seems to have little or no direct influence on prognosis. Pregnancy certainly entails risks.

**Civil state.**—In active cases marriage affects the outlook unfavourably. In arrested cases breakdown not infrequently occurs shortly after marriage.

**Physique.**—A poor physique is usually of bad men, whereas a good physique is, other things being equal, favourable.

A persistent loss of weight in spite of treatment is a most unfavourable sign.

**Personality** is of great importance. Austin Flint stated that "most patients who recover from phthisis are persons of resolution and perseverance."

**Type of disease.**—This influences prognosis, and may be seen by reference to the section on the course of the disease.

**Symptoms and prognosis.** Persistent cough may lead to exhaustion and be unfavourable. Hemoptysis may in rare instances be immediately fatal or lead to death from exhaustion in a few days. It may also cause wide dissemination of the disease from inhalation of sputa into healthy parts of the lung. On the other hand repeated hæmorrhages may occur in the course of years without ill effects. Dyspnoea of serious import, while fever and night sweats indicate activity. Digestive symptoms are a handicap at any stage, and if persistent are unfavourable. A blood-pressure of 100 mm. or less is ominous, while a rising pressure is favourable.

The extent of physical signs is only of significance when taken in conjunction with the symptoms as an index of activity. The rapid development of fresh signs is necessarily serious.

**Complications and prognosis.**—*Laryngeal involvement* is not invariably so serious as was formerly supposed, if the lung lesions are localized and quiescent and if proper treatment is carried out. *Pneumothorax* is serious. Often it is rapidly fatal, but, even if not, final recovery is rare. *Pleural effusion* may exert a favourable influence by keeping the lung at rest. *Catarrhal infections* are unfavourable, serving to spread the disease or to activate quiet lesions. *Meningitis* is almost invariably fatal. *Intestinal tuberculosis* is of evil omen, and may lead to exhaustion or perforation.

*Genito-urinary tuberculosis* is serious, but if localized to the epididymis may be dealt with by operation.

### TREATMENT

The selection of the treatment must depend upon the type and stage of the disease and the patient's pecuniary circumstances. In the febrile stage, or when acute symptoms are present, the treatment required is closely similar to that for other acute febrile illnesses, and for such treatment the home, a nursing home, or a hospital may be the best place. It is only when the patient becomes afebrile that some of the special modes of treatment become possible. In chronic cases variations in the treatment are often necessary.

**1. Sanatorium treatment.**—The rôle of sanatorium treatment in the general scheme of dealing with tuberculosis, the principles on which it is based, and the selection of suitable cases are discussed in the general article on Tuberculosis.

The buildings of a sanatorium should be designed so that the rooms face south or south-west. There is generally a paved and covered space in front so that patients confined to bed can be out under shelter. Common rooms for meals and amusements are provided. Sometimes chalets or revolving shelters are used instead of permanent buildings. Carpets are to be avoided, though a small rug may be allowed. Furniture should be simple, and as little as is consistent with comfort. The grounds should be large enough to permit of varying walks and afford facilities for graduated work. The personality of the medical superintendent is an important factor.

It is usual for patients arriving at a sanatorium to be kept in bed for a few days so that



## PULMONARY TUBERCULOSIS

they may recover from the journey, and that the resting temperatures may be recorded. Patients with a febrile reaction are kept in bed till this has subsided. The daily routine differs according to the condition of the patient, the stage of the disease, and the form of special treatment selected.

The ordinary daily routine is somewhat as follows :—

7.30 A.M.—Patient called. Temperature and sometimes pulse-rate taken—bath if permitted, then dress.

8.30 A.M.—Breakfast.

9 to 9.30 A.M.—Rest, reading, or mild recreation.

9.30 to 12 noon.—Patients are generally seen by the medical officer at some time between 7.30 and 9.30, and the day's programme is given to them. It may be rest, a prescribed walk of given length with a stated rate, or a specific grade of "labour."

12 to 1 P.M.—The rest hour, strictly enforced for all except convalescents. The patient rests on a couch or bed on the veranda, in a shelter or out of doors. No speaking, writing, or working is permitted, but reading is usually allowed.

1 P.M.—Dinner.

After dinner till 2.30.—Rest, reading, and mild recreation are again permitted.

2.30 to 4.30.—Rest, exercise, or work, as prescribed for each patient.

4.30 P.M.—Tea. After this till 6 P.M., rest, letter-writing, or mild recreation again.

6 to 7.—Second rest hour, under conditions similar to the first.

7 or 7.15.—Supper. Again gentle recreation, such as music, singing, or games like chess and draughts, is permitted till 9.

At 9 or 9.30 P.M. patients go to bed.

The rest hours are a very important part of the treatment; they prevent fatigue, allow the exercise temperatures to subside, and prepare for digestion.

The duration of stay in the sanatorium should not be less than from three to six months. Few patients can afford to remain until arrest is complete, but the experience gained enables them to carry out treatment at home or at suitable health resorts.

**2. Home treatment.**—This may be necessary (1) in the early or the febrile stages, (2) after return from a sanatorium or climatic cure, (3) in the late or terminal stage, though in this stage it is desirable that the patient should be admitted to some institution for advanced cases unless home conditions permit of adequate nursing and due precautions.

It is important to secure conditions of life as

nearly as possible approximating to those of a sanatorium. The patient should have a room with a south or south-west aspect, all unnecessary furniture, carpets, curtains or dust-retaining features should be removed and the bed be so placed as to ensure the maximum of fresh air without draughts, windows being permanently opened and if necessary partly removed.

The *diet* should be adjusted to the patient's digestive capacity and personal tastes. Since one object is to restore him to his "highest known weight," it is often necessary to encourage the taking of extra food, particularly fats and proteins, but it is not desirable to enforce the large extra feeding at one time recommended. A diet with a heat value ranging from 3,000 to 3,500 Calories is ample in the majority of cases, though it may be necessary to increase it to 4,000 in some instances. The protein may be given in the form of meat, fish, and eggs. Raw meat has been strongly recommended, and may take the form of sandwiches suitably flavoured. The meat may be slightly browned before the fire to lessen the raw taste, and as much as half a pound daily may be given. The amount of cooked meat should not exceed  $\frac{3}{4}$  lb. a day; if fish or eggs are included in the diet, that amount may be proportionately diminished. Carbohydrates may be increased, particularly if the patient is on active exercise or is losing weight.

Fats are generally well tolerated by tuberculous patients, and may be given up to 4–6 oz. a day in the form of milk, butter, cream, fat of meat, fat bacon, or cream cheese.

Large quantities of milk are sometimes enjoined, but if the patient is on a mixed diet one to three pints is sufficient.

Alcohol is not necessary, but may be permitted in moderation at meals in those accustomed to its use.

**Hygiene.**—Smoking tends to induce coughing and should not be indulged in when there is active disease, fever, or laryngeal involvement, but may be permitted in quiescent cases.

Clothing should be adapted to the climate, the season, and the condition of the patient. Good woollen underwear, with clothes of suitable weight, and overcoats or rugs according to the weather, should be worn, but excess of clothing is to be avoided.

Strict care in regard to coughing and the disposal of sputum should be enjoined. Patients with expectoration should be advised to shave. Sputum can be collected in a special pocket

## PULMONARY TUBERCULOSIS

flask, in paper or gauze handkerchiefs, or in sputum cups from patients in bed. It should be treated with some cheap disinfectant, such as crude carbolic acid, weak lysol or sanitas, or strong solution of washing soda, and afterwards burned if possible.

**3. Climatic treatment.**—Although of great value in some cases, this form of treatment is not available for the great majority and is unsuitable for many. It is discussed in CLIMATE IN THERAPEUTICS.

**4. Graduated rest and exercise.**—Rest in bed is essential for patients with fever, and if this is slow to subside it must be made "absolute," i.e. the patient must not get out of bed, must be washed and fed, and must use the bed-pan for evacuations. When the temperature has subsided, "absolute" rest may be relaxed, and, if the temperature keeps down, the patient may be allowed up for an hour daily, then two hours and so on, the temperature chart being the guide. When the patient is up for from four to six hours a day, gentle exercise may be permitted, and gradually walks may be allowed. To get to this stage may require weeks or months, and it is often a great tax on his forbearance. As the disease progresses towards quiescence and arrest, longer walks at steeper gradients become possible, or the patient may be put on some form of graduated labour. As introduced by Paterson at Frimley Sanatorium, various grades of work, from one to six, are employed. The first grade is started when the patient can walk six miles a day, and consists in carrying a light weight uphill; the other grades involve increasing work till in the sixth grade it becomes heavy labour with a pickaxe or spade for six hours daily. The patient must be carefully watched for any indication of auto-inoculation, and at once put to rest if this occurs. One advantage of this mode of treatment is that it is a better preparation for the patient's resumption of his ordinary work than graduated walking.

**5. Medicinal measures.**—The mere list of drugs recommended for this disease is the best proof of their general inefficacy. Some of the more important may briefly be discussed.

*Creosote* has been widely used, and seems sometimes to do good. It is best given in perles or capsules starting with 2 min. three times a day after meals and increasing gradually. It should be stopped if it induces dyspepsia or if hæmoptysis occurs. Various derivatives and compounds of creosote have been used, such as guaiacol 1 3 min., creosote carbonate (creo-

sotal) 5–10 min., guaiacol carbonate 10–30 gr., guaiacol cinnamate (styracol) 5–15 gr., potassium guaiacol sulphonate (thiocol) 5–15 gr.

"Nascent" iodine has been recommended; the patient takes 30 gr. of potassium iodide with or without potassium bicarbonate in at least half a pint of water after breakfast, and chlorine water with lemonade is given at intervals during the day to a total quantity of 3–5 oz. This treatment seems sometimes to help chronic cases, but is often disappointing and may disturb digestion.

The *hypophosphites* formerly had a great vogue. They may help the general nutrition and well-being of the patient. Like the glycerophosphates, they are often combined with bases such as calcium, magnesium, and iron to combat the supposed demineralization induced by this disease. Fresh bone powder has been suggested for the same purpose.

Various *arsenical preparations* have been recommended, among them liquor arsenicalis, 2–3 min. by mouth, sodium cacodylate hypodermically in doses of  $\frac{1}{4}$ – $\frac{1}{2}$  gr. daily, in fortnightly periods, with intervals, or salvarsan intravenously. Salvarsan is useful in cases in syphilitic subjects, but is not devoid of danger.

*Succinimide of mercury*,  $\frac{1}{2}$  gr. in 10 min. of water hypodermically every other day till thirty doses have been given, is also used, especially in cases with sv; hilis.

*Cod-liver oil* has long had a great reputation and may be given up to 2 oz. daily. Recently Sir Leonard Rogers has suggested the use of *sodium morrhuate*. A 3-per-cent. sterilized solution with  $\frac{1}{2}$  per cent. of phenol is given at first hypodermically, then intravenously. Doses of  $\frac{1}{2}$  c.c., increased up to 2 c.c., are given two or three times a week till febrile reaction occurs.

*Garlic* and *allyl sulphide* have been recommended.

Attempts have been made to treat the lung lesions by *antiseptic inhalations*, but, though benefit may accrue from them, they are not specifically curative. They are best given on a Yeo respirator, worn for as many hours daily as possible. The best-known is that recommended by D. B. Lees for continuous inhalation, viz. creosote 2, carbolic acid 2, tincture of iodine (mild) 1, spirit of ether 1, spirit of chloroform 2. Of this 6 to 8 drops per hour are put on the sponge of the respirator. A modification in use at the Brompton Hospital is menthol 4, oil of cinnamon 3, oil of lemon 4, creosote 20, oil of pine 10, spirit of chloroform 10.

## PULMONARY TUBERCULOSIS

These inhalations frequently lessen cough, reduce expectoration and give comfort to the patient.

**6. Specific methods.** (1) **Tuberculin.**—Opinions differ widely as to the usefulness of tuberculin in pulmonary tuberculosis. It has been extensively used in different forms and in various dosages.

The forms of tuberculin fall into three groups, and are characterized by certain initials: (1) The *exotoxins*, or soluble metabolic products of the bacillus—e.g. Koch's original tuberculin T., O.T., or T.O.A., Denys's bouillon filtré B.F., and albumose-free tuberculin A.F. (2) The *endotoxins* or insoluble chemical constituents of the bacterial bodies, e.g. Koch's new tuberculin T.R. (3) *Mixtures of the exotoxins and endotoxins*, such as Koch's bacillary emulsion B.E., Béraneck's tuberculin T.Bk., or sensitized bacillary emulsion S.B.E.

Tuberculin may be made from either human or bovine bacilli, the latter forms being distinguished by the letter P. (Perlsucht), e.g. P.T., P.T.R., P.B.E. Autogenous tuberculins may be used.

It would seem that in general the soluble forms are quicker and more likely to induce reactions, the tuberculins of the second and third groups being slower and milder. The form is of less importance than the method of administration and dosage. There are two methods, one in which reactions are expected and utilized, involving the use of increasing doses, at intervals of from three to ten days, with the view of producing tuberculin tolerance, the other in which doses are given at longer intervals, reactions being avoided as far as possible. The doses are either graduated in milligrammes as originally recommended by Koch, or in cubic centimetres or millimetres, thus giving rise to considerable confusion. The method employed by Riviere and Morland of using the cubic millimetre in dosage has much to commend it. The necessary dilutions are easily made by means of a series of sterile bottles, preferably amber-coloured, in each of which is put 9 c.c. of a 0·8-per-cent. sterile solution of sodium chloride containing 0·5 per cent. of carbolic acid. To the first, 1 c.c. of the original fluid obtained from the makers is added. After mixing, 1 c.c. of this is added to the second, and so on. The sixth bottle will give a 1-in-a-million dilution, and 1 c.c. will equal 0·001 c.mm. of the original fluid. Such a dose would equal 1/1,000 mg. of T., P.T., B.E., or A.F., 1/100,000 mg. of T.R., or 1/500,000 mg. of B.E.

The initial dose varies, some authorities starting with minute doses such as those just mentioned, while others begin with much larger ones, even as much as 0·5 to 1 c.mm. The final dose is also much debated, some authorities going on to as much as 1 c.c. of the original fluid, while others stop at one-tenth of this dose.

The method of administration is by simple subcutaneous injection with ordinary aseptic precautions. The temperature should be carefully recorded and any subjective, local or focal manifestation observed.

*Uses of tuberculin in treatment.*—The enthusiasts for tuberculin regard most cases as suitable except patients with advanced disease, those with fever and rapid pulse, and those complicated by secondary infections or intercurrent disease. Other observers regard it as of doubtful utility and either discard it altogether or limit its use to chronic afebrile cases. The writer is more convinced of its power for harm in pulmonary cases if injudiciously employed, than of its usefulness, though he thinks that if given cautiously without inducing reactions it may assist in promoting fibrosis and in getting rid of bacilli from the sputum in chronic cases progressing towards arrest.

(2) **Passive immunization methods.**—The anti-tuberculosis serums prepared by Maragliano and Marmorek have not established the claims of their authors and are but little used in this country. Mehnarto's contratoxin and Spengler's I.K. have likewise had a limited vogue and been discarded by the majority of workers here.

**7. Operative measures.** (1) **Artificial pneumothorax** has now established its value as a mode of treatment for certain cases. These are in the main patients with advancing disease associated with fever and toxic symptoms, in whom the active lesions are practically unilateral. It is also sometimes beneficial in cases with severe or repeated hæmorrhages. It should not be employed in early cases, in those progressing favourably, or in those with extensive signs on both sides. It is also inadvisable in cases with severe cardiac enfeeblement, and impracticable in fibroid disease with old pleural adhesion. Laryngeal involvement and even intestinal tuberculosis do not necessarily contraindicate its use. It has therefore a limited field of application, but its special value is that it is beneficial in cases which would otherwise progress unfavourably.

The principle of the method is to introduce

## PULMONARY TUBERCULOSIS

sterile air or nitrogen into the pleural cavity, producing collapse of the affected lung and keeping it at rest by repeated refills. In successful cases the temperature rapidly falls to normal ranges, cough and expectoration diminish, and the patient is enabled to be up and take exercise and even to work.

The apparatus required consists of (1) a special needle such as that introduced by Saugman; (2) two bottles, one containing the nitrogen or air, the other containing water, the two being connected by rubber tubing and so arranged that water can flow down and displace the gas; (3) a water manometer; (4) a three-way stopcock and tubing. Convenient forms of apparatus, such as Lillingston's (Fig. 84) are on the market.

**Technique.**—The patient should be in bed recumbent, or slightly inclining toward the sound side, and an injection of morphine is advisable for the first induction. The spot chosen for injection should be at a distance from any excavation and at a spot where there are no indications of pleural adhesion. A convenient place is the axilla in the sixth to the ninth intercostal space. The skin is cleaned and painted with iodine, and novocain is injected, at first below the skin, then deeper till the pleura is reached. For the first induction Clive Riviere's needle is to be preferred. After sterilization it is to be dried, then attached to the apparatus and carefully inserted with the sharp trocar *in situ*. When the pleura is reached, the latter is withdrawn and the blunt cannula pushed through the parietal pleura, an incident which can usually be recognized. The stopcock is then turned to connect the manometer. If the needle is still in the muscles, the readings remain at zero; if it is in contact with the pleura, slight oscillation may be obtained. If the needle has gone through the parietal pleura, a negative pressure of 5–10 c.c. with respiratory oscillations is observed, and no nitrogen should be introduced till this is established. If there are adhesions preventing the entrance of the needle into the pleural space, the manometer will remain at zero, or only show slight oscillations; so also if the needle has entered the lung. The utmost care should be taken to make certain that the needle is in the pleural space before introducing the air or nitrogen, and for this the manometer reading is the best guide. For a first injection a maximum of 500 c.c. should be introduced, except in cases with hæmorrhage, in which 1,000 c.c. may be necessary. If adhesions are present, only 100–200 c.c. may be possible. The

first refill may be necessary next day in hæmorrhagic cases, or on the third day in others, and the interval is gradually increased to a week, then a fortnight, and eventually to four or six weeks, the quantities of gas being increased to 600–700 c.c. when possible. It will be necessary to continue the refills for eighteen months or longer, and no definite rule can be given, since it depends upon the circumstances of each case.

The chief risks of the method are with the

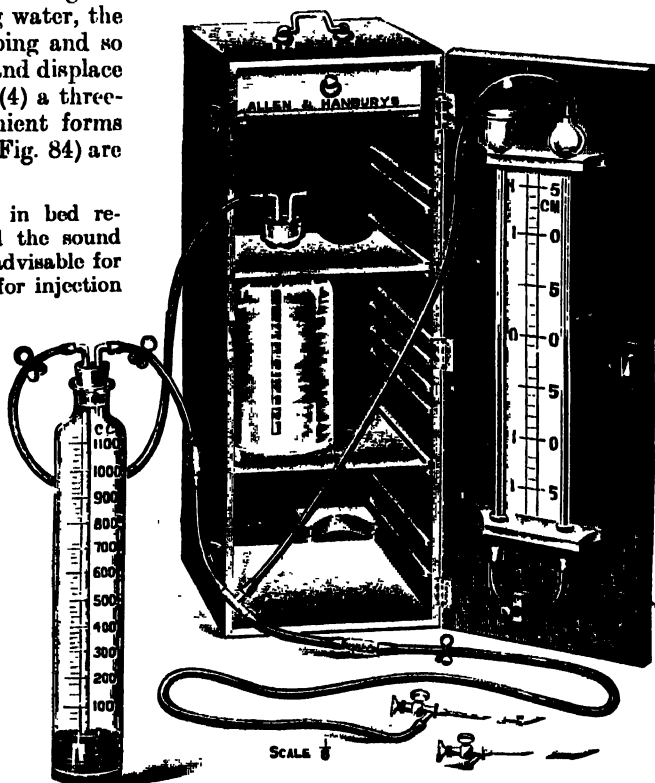


Fig. 84.—Lillingston's apparatus for artificial pneumothorax.

first injection, and comprise pleural shock and gas embolism, either of which may be fatal; but with due precautions these risks are small. The method may fail because it may be impossible to induce collapse owing to adhesions, or because the other lung becomes more involved. Pleural effusion, pyo-pneumothorax, spontaneous pneumothorax from puncture of the lung, and subcutaneous emphysema may occur as complications.

(2) **Thoracoplasty and other operations.** When artificial pneumothorax fails to induce collapse of the lung, the operation of thoracoplasty may be considered, provided the general condi-

## PULMONARY TUBERCULOSIS

tion of the patient is satisfactory. It has been employed by Saugman and other Scandinavian observers with considerable success. It can be done under local anæsthesia, and is probably best performed in two stages, parts of the lower ribs posteriorly being removed at the first, the upper ribs being dealt with at the second. Rib mobilization, or Wilm's operation, is also performed in two stages, and here parts of the ribs are removed both in front and at the back. Section of the phrenic nerve is sometimes carried out to induce basic collapse.

These operations involve permanent collapse of the whole or part of one lung, and should only be considered when artificial pneumothorax has failed or is impracticable.

8. **Symptomatic treatment.**—Though indiscriminate administration of drugs is to be deprecated, the judicious treatment of symptoms may contribute to the patient's comfort, particularly in unfavourable cases.

*Cough.*—A useless, ineffective, or painful cough may be checked by a linctus or sedative lozenges containing substances such as heroin, codeine or liquorice. The tenacious sputum of the early morning may be loosened and vomiting often prevented by a mixture consisting of sodium bicarbonate 10 gr., sodium chloride 3 gr., spirit of chloroform 10 min., anise water to 1 oz., or by a cup of hot milk and water. When there is sputum the cough should not be interfered with, unless it be to give a sedative at night to ensure sleep. If the expectoration is offensive, antiseptic inhalations may be employed, or garlic or allyl sulphide given internally.

*Dyspnœa* can only be helped by treating any mechanical condition causing it, such as a pleural effusion. Oxygen is rarely needed, except to relieve distress in the terminal stages.

*Pain* can sometimes be alleviated by local counter-irritation through liniments, painting with tincture of iodine, the application of a mustard leaf, etc. If it is due to dry pleurisy, strapping has been recommended, but in some cases it may lead to dyspnœa.

*Night sweats.*—The bedroom should be well ventilated. The patient should have a second night suit to change into after he has been dried. He may be sponged before going to sleep with toilet vinegar and water. Various drugs have been recommended, among them zinc oxide or valerianate 3-5 gr., dry extract of belladonna  $\frac{1}{2}$  gr., atropine  $\frac{1}{32}$  gr., picrotoxin  $\frac{1}{80}$  gr., agaricin  $\frac{1}{40}$  gr.

*Fever.*—The treatment for fever is rest in bed, absolute if it is high or intractable, partial if it is of moderate degree. If the temperature does not exceed 99.5° F., the patient may be allowed up with caution and the effect on the chart watched. Antipyretics are sometimes given, and may afford some relief to the symptoms, but they have no beneficial influence on the disease and are best avoided if possible. Among those employed are quinine 3-5 gr., aspirin 5-7 gr., pyramidon 5-10 gr., and cryogenin 7-10 gr. in cachet.

*Hæmoptysis.*—Treatment depends on the degree. If it occurs in streaks only, no special treatment is necessary. If it amounts only to one or two ounces, rest in bed, an aperient and some sedative to calm the patient may suffice. If it is profuse or persistent, he should keep strictly to bed and lie on the affected side if possible. A small injection of morphine  $\frac{1}{4}$  to  $\frac{1}{2}$  gr., or heroin  $\frac{1}{16}$  to  $\frac{1}{8}$  gr., may lessen the cough, soothe the patient, and quieten the heart. Inhalation of amyl nitrite 3-5 min. is sometimes used and may be helpful. Ice is often sucked, but if much is used it induces flatulence. A saline aperient, preferably sodium sulphate, should be administered if there is constipation. Calcium lactate, 10 gr. three times a day, is often prescribed for two or three days. Injections of horse-serum or "coagulose" appear to be helpful in some cases. Adrenalin and ergot should not be used. Dilute sulphuric acid or gallic acid has been given in mixtures, apparently with benefit.

Daily injections of emetine hydrochloride  $\frac{1}{4}$  gr. seem to help in cases of repeated small hæmorrhages. In any severe recurrent hæmoptysis, artificial pneumothorax may be considered. In all cases of severe bleeding, the food given should be cool, and alcohol forbidden. Recently, some authorities have urged that the usual restrictions as to activity imposed on these patients are unnecessary, and others strongly deprecate the use of morphine. There is certainly a tendency to keep patients in bed longer than is necessary.

*Digestive symptoms.*—Anorexia may be relieved by an alteration of the régime, and by change of diet. Dyspeptic symptoms should be treated on general lines. Alkalis, bitters, takadiastase, papain, and gentian are often useful in combination. Constipation can be dealt with by diet, fruit, or laxatives, as may be necessary. Diarrhœa is often troublesome, and in the first instance the treatment should be dietetic, avoiding coarse or fibrous foods.

## PURPURA

Later, bismuth, opium, tannigen, salol, or  $\beta$ -naphthol may be tried.

*Cardiac enfeeblement* can be treated by drugs such as digitalis, strophanthus, nux vomica, and caffeine.

*Insomnia* is treated on general principles. Useless cough is checked, digestive symptoms are treated and, if necessary, small doses of hypnotics are given.

The treatment of complications such as pleural effusion, empyema, pneumothorax, bronchiectasis, and pericarditis follows the ordinary methods appropriate to each.

Questions connected with the after-care of patients are discussed in the general article on Tuberculosis.

R. A. YOUNG.

**PULSATING AORTA** (*see* AORTA, DYNAMIC PULSATION OF).

**PULSATING EXOPHTHALMOS** (*see* ORBIT, AFFECTIONS OF).

**PULSE, ABNORMALITIES OF** (*see* HEART-BEAT, ABNORMALITIES OF).

**PUPILS, ABNORMALITIES OF** (*see* OPHTHALMOPLÉGIA).

**PUPILS, EXAMINATION OF** (*see* EYE, EXAMINATION OF).

**PURPURA.**—A condition characterized by the occurrence of spontaneous hæmorrhages into skin and mucous membranes.

**Etiology and pathology.**—The skin lesions result from vascular rupture, commonly of the subdermic venules. In a few instances organisms have been isolated from the blood. Wright has demonstrated that the coagulation time of the blood may be delayed. Purpura is more frequently a symptom, occurring in many diverse conditions, than an idiopathic disease.

**Symptomatology.**—On the skin, flat or slightly elevated spots, from a pinhead (petechiæ) to large areas (ecchymoses) are seen; these lesions alter in colour from red to green or yellow. Urticaria, erythemata, and even bullæ may be associated with the true purpuric eruption. The disease often lasts over long periods, the lesions coming out in crops, and as a rule causing no discomfort except on the lower limbs, where itching may be felt. Purpura is divided into *primary* or idiopathic, and *secondary* or symptomatic.

**Primary purpura.**—(a) *P. simplex* is the mildest form of the primary disease, and is commonly seen in children, the rash being

usually the only evidence of disease, although joint-pains and diarrhœa sometimes occur. It may continue irregularly for months or even years (*P. chronica*).

(b) *P. hæmorrhagica* (Werlhof's Disease) may be regarded as a severe form of the above. The onset is sudden, with high temperature and bleedings from mucous membranes; even apoplexy may occur. Both skin and mucous membranes are involved, and the lesions may be widely spread. Young, delicate girls are especially liable to attack. A fatal variety (*P. fulminans*), in which death occurs within twenty-four hours, is rare.

(c) *P. rheumatica* (Schönlein's Disease) is a combination of arthritic symptoms with purpura, and is sometimes called *arthritic purpura*. The joint-swellings are usually slight; rarely there is endocarditis. Wheals and erythemas of the multiform or nodose types often complicate the eruption. This variety, which especially affects males of from 20 to 30, may begin with sore throat and fever; occasionally there is albuminuria; relapses and recurrences are common.

(d) In *Henoch's purpura* also both purpuric spots and urticarial wheals may be present. So intense is the process that bleeding takes place from bowel and mouth; in consequence of hæmorrhage into the wall of the gut, symptoms resembling intussusception sometimes occur. Joint-swelling, and nephritis may be met with. The mucous membranes are usually involved.

**Secondary purpura.**—In these cases the purpura is associated with some recognized disease or abnormal condition, such as a malignant fever, sepsis, rheumatism, toxic states from drugs (especially potassium iodide), renal, hepatic, or cardiac disease, whooping-cough, or epilepsy; blood diseases and wasting conditions like cancer or tuberculosis must also be included.

**Diagnosis** is usually easy, the purpuric spots remaining on pressure; but flea-bites are sometimes confusing. It is somewhat less easy to decide whether the purpura is primary or secondary. The primary groups, however, are fairly definite, and the only one which is really difficult to recognize is Henoch's purpura, which may closely simulate intussusception. All the conditions responsible for secondary purpura should be sought for by a systematic examination of chest, abdomen (spleen), blood, and urine, while a high temperature will point to the presence of some septic state. Common

## PYELOGRAPHY

causes of secondary purpura are renal disease, infective endocarditis, and "cachectic" states.

**Prognosis** should always be guarded, as even a mild variety may change into a severe type. In the secondary forms the prognosis depends on the causal condition.

**Treatment.**—Every case must be treated seriously, and the patient should be ordered to bed. Internally, oil of turpentine may be given in 5–10-min. doses in emulsion; in malarial cases quinine, in rheumatic cases salicylates, are indicated, while calcium lactate (20 gr. every four hours) should be given when blood coagulation is slow, but should be discontinued after three or four days. Both sulphuric acid and tincture of iron are most useful. The general health should be attended to, and any oral sepsis corrected. In severe cases ergot hypodermically, horse-serum (10 c.c. subcutaneously), or physiological saline (100 c.c. subcutaneously) may be tried, while ice may be administered internally or applied locally to check the bleeding.

H. MACCORMAC.

**PUTREFACTION** (see POST-MORTEM EXAMINATIONS IN MEDICO-LEGAL CASES).

**PYÆMIA** (see SEPTICÆMIA AND PYÆMIA).

**PYELITIS** (see PYELONEPHRITIS AND PYELITIS).

**PYELOGRAPHY.**—This process consists in obtaining an X-ray shadow of the renal pelvis and calyces, which have been filled with an opaque fluid introduced through a ureteric catheter. The examination is made in the radiographer's room. The patient lies on a radiographic couch, and cystoscopy is performed in the usual way with a catheterizing cystoscope. A catheter opaque to the X-rays is passed into one or both ureters, and through this the opaque medium is introduced into the renal pelvis. The catheter should if possible be passed into the renal pelvis, but various causes may prevent it passing the full length of the ureter. In this case it will be found that so long as there is no regurgitation into the bladder from the eye being too close to the ureteric meatus, the fluid will ascend and fill the renal pelvis. When the catheter is in position the cystoscope is withdrawn, and sodium bromide solution, previously warmed, is introduced in the following manner: The solution is drawn into the barrel of an all-glass syringe of 20 c.c. capacity, the needle of which is inserted into the end of the ureteric catheter. The syringe

is now raised vertically to about 6 or 8 in., and, by the aid of a small electric light held behind it, the column of fluid is seen to fall.

Two signs indicate that the renal pelvis is full and the introduction must cease—pain in the renal pelvis, and hesitation and cessation in the descent of the column of fluid in the syringe. When the pelvis is filled, the catheter is plugged and the exposure to the X-rays made without any delay. The fluid is then allowed to run off, the renal pelvis gently washed with saline solution, and the catheter removed. No general or local anæsthetic should be used to prevent pain in the renal pelvis, for in this way an important guide would be lost.

The solutions suitable for use are sodium bromide solution (20 per cent.), collargol (5 or 10 per cent. strength), and thorium nitrate (15 per cent.). Of these, sodium bromide solution is the least irritating and the most convenient. The shadow is very slightly less heavy than that of collargol.

Along certain lines this method has a very important use as an aid to diagnosis by providing information which is vital to the diagnosis and treatment of the case, but which cannot be obtained by other and more simple methods. Such information relates to—

1. Early diagnosis of *dilatation of the kidney* in the stage when a swelling cannot yet be detected in the abdomen and before there is extensive destruction of kidney tissue. These may be cases of calculus in the renal pelvis or ureter, or of movable kidney, or the hydronephrosis may be due to an aberrant renal vessel or other abnormality at the upper end of the ureter.

2. The *localization of supposed stone-shadows* in the renal area. In some cases a shadow which falls within the renal area may be proved to be extrarenal, in others a stone-shadow can be accurately localized.

3. The *diagnosis of abdominal tumours*. It is in the cases of abdominal tumours in which there are no localizing symptoms such as hæmaturia, melæna, leucocytosis, or jaundice, or local character such as a sharp notched margin, that the method is useful. I apply an opaque paint to the area of the tumour on the surface of the abdomen, and the relation of this to the opaque renal pelvis and calyces assists the diagnosis.

The dangers of the method are irritation of the renal pelvis with the solution, forcing the solution into the renal tubules, and even rupture of these or rupture of a hydronephrotic

## PYELONEPHRITIS AND PYELITIS

kidney. Necrosis of areas of the kidney has been described. These dangers may be avoided by delicacy of manipulation and careful attention to detail in the technique. The method is only suitable for use by an expert in cystoscopy and ureteric catheterization.

J. W. THOMSON WALKER.

### PYELONEPHRITIS AND PYELITIS.—

An inflammation of the kidney involving both the renal parenchyma and the pelvis. *Pyelitis* signifies an affection of the pelvis alone, but as this never occurs without some involvement of the secreting substance, the two terms are synonymous for all practical purposes. Some chemical substances, such as cantharides, turpentine, copaiba, etc., when excreted in large quantities by the kidney, act as an intense irritant to that organ, but, with this exception, pyelonephritis is always of bacterial origin.

#### 1. PRIMARY ACUTE PYELONEPHRITIS

By primary acute pyelonephritis is meant a sudden invasion by pathogenic organisms of an apparently healthy kidney. The infection is usually by a bacillus of the coliform group, and it is to these cases that the following description mainly applies. In certain cases pure cultures of staphylococci, streptococci, or proteus are obtained from the urine, but their symptoms are so similar and their occurrence so infrequent that they do not warrant a special description.

**Etiology.**—In many *B. coli* infections of the urinary tract the symptoms may, for a time, be slight or even absent, so that it may be impossible to be certain of the exact site of the initial invasion. Moreover, although attention is often directed to the condition by symptoms of bladder irritability, such as increased frequency or lack of control, it does not follow that the original trouble was situated in that viscus. Cystoscopic examination of the ureteric orifices at the time of onset of symptoms may point clearly to disease, already of some duration, in one or both kidneys.

The disease is commonest in women and female children. Many of these cases are associated with some disorder of the intestine; constipation is the commonest, but in infants under 2 years the affection is very apt to follow an acute attack of diarrhoea. In children the right kidney is affected three times as often as the left; in women a still greater preponderance of the cases occur on the right side. If the affection be a bilateral one,

the onset is rarely simultaneous on the two sides, and the stress of the disease almost invariably falls upon the right kidney.

It is uncertain whether the organisms reach the kidney by direct invasion from the colon or whether the infection is a blood-borne one. Cases described as "pyelitis of pregnancy" differ in no essential respect from those referred to above. In them the cause predisposing to infection is renal stasis produced by pressure of the enlarged uterus on the ureter, where the latter enters the pelvis. When the source of the obstruction is removed by the emptying of the uterus, either naturally or otherwise, these patients quickly recover.

**Pathology.**—The appearances seen in the kidney depend upon the stage to which the disease has progressed before resolution occurs, and upon the virulence of the infecting organisms. In the slighter cases the kidney is enlarged and engorged with blood, and its microscopical characters are very similar to those seen in the nephritis of some of the acute specific fevers. The kidney recovers in a great measure, if not completely, and, except for an increased liability to a second attack, may be regarded as a normal organ. In the more severe infections suppuration occurs, and small abscesses and areas of necrosis are found scattered throughout the cortex and medulla. The kidneys, however, appear to be possessed of considerable recuperative power, and some of the atrophied and scarred organs seen in the post-mortem room have undoubtedly arisen from a condition such as this.

In less favourable cases the kidney gradually becomes the seat of a chronic pyonephrosis, but there is not necessarily any great enlargement of the organ.

**Symptomatology.** (a) *Simple cases.* The attack begins with acute pain in the front and back of one or both loins, nausea, and fever, the temperature quickly rising to 103° F. or even higher. There is much rigidity with tenderness over the region of the affected kidney, which, although enlarged, is seldom palpable. Within a few hours of the onset, micturition becomes very frequent, and much gripping pain is experienced in the region of the bladder neck; the symptoms of bladder irritability may actually precede those attributable to the renal condition. Hæmaturia may occur, but is unusual. The urine for the first twenty-four hours may show no change, but it soon assumes a hazy opalescent appearance, and on examination is found to be swarming with organisms



## PYELONEPHRITIS AND PYELITIS

and to contain a certain number of pus cells. It may be acid or neutral in reaction, and is not necessarily offensive. The quantity of pus varies from a microscopic amount to a quite copious deposit. There is usually a distinct cloud of albumin on boiling. The attack is usually at its worst during the first thirty-six hours of the disease; the pain and fever then begin to abate, the urine gradually clears, and within five or six days of the onset the patient to all intents and purposes is quite well. The bacilluria unfortunately persists for a considerable time, and it may need many months of careful treatment before the urine proves sterile on cultivation and the patient can be considered free from the risk of recurrence.

(b) **Suppurative cases.**—In those cases which progress to suppuration the symptoms are very similar, and differ only in their much greater severity and longer duration. After a short preliminary period of general malaise and headache the attack often begins with a well-marked rigor, which may subsequently be repeated daily, accompanied by very acute pain in the loin and back, and vomiting. There is much sweating with collapse, and the patient looks and feels extremely ill. The temperature rises rapidly to 105° F., 106° F., or even higher, and, though subject to considerable oscillation during the first few weeks of the illness, rarely actually reaches the base line in its excursions. Although there is considerable muscular rigidity over the affected loin, after the first few days it is usually possible to feel an enlarged and tender kidney. The spleen is often considerably enlarged also, and, together with the affected kidney, may alter in size from day to day. The urine is diminished in amount, and its characters are subject to considerable variation. Bursts of pyuria are accompanied by remissions in temperature and a general abatement of symptoms, and alternate with periods in which almost clear urine is passed and a recrudescence of the pain and fever occurs. The attack usually lasts from two to four weeks, during which period there is great distaste for food and the patient wastes rapidly. With the disappearance of active symptoms the pus in the urine begins to diminish, the general condition to improve, and after a few weeks the patient may be regarded as on the way to convalescence. The bacilluria may persist for six months or even longer. In the less favourable cases death may occur from septicæmia during the first week of the illness. In others, relapse may follow relapse, and, in

bilateral cases, the patient may gradually waste away from prolonged renal suppuration and eventually die of uræmia.

**Diagnosis.**—The symptoms of acute pyelonephritis may simulate closely those due to some acute peritoneal condition such as a *perforated gastric ulcer* or *acute appendicitis*, and may easily be mistaken for them. A correct diagnosis can usually be made by noting the following points:

(1) The muscular rigidity and tenderness are confined to one or both loins. The tenderness is often particularly noticeable in the postrenal angle.

(2) The extremely high temperature, which is out of proportion to the rise in the pulse-rate.

(3) The presence of symptoms of bladder irritability.

(4) The condition of the urine.

**Treatment.**—Simple cases do not require any active surgical treatment, because the natural tendency is to recovery. The patients should be confined to bed, placed on a milk diet, and encouraged to drink large quantities of barley water, whey, and citrated milk. Alkalis (pot. cit. 30 gr.) are especially useful in children, and may with advantage be combined with 5-gr. doses of urotropine. Pain should be relieved by hot fomentations to the abdomen and, if necessary, by suppositories of belladonna and morphia. The motions are often very offensive, and careful attention should be directed to the proper regulation of the bowels. Autogenous vaccines are sometimes followed by much improvement in the symptoms and temperature chart, but do not appear to cut short the disease or to have any influence on the bacilluria. After the acute symptoms have subsided the patient should, if possible, be removed to a sunny, warm climate for several months and placed under the best hygienic conditions. Salol, given in 10-gr. doses thrice daily, appears to be by far the most efficient antiseptic.

In the **suppurative** cases immediate operation is only required in exceptional circumstances. A few of the most virulent infections are fatal within the first week, from septicæmia, but as a very large proportion of these cases recover without operation, and as there is no means of identifying this particular group before a general infection has occurred, nephrectomy is seldom called for at this stage even if the disease is limited to one kidney.

In some *unilateral* cases, however, when, after three or four weeks of continuous fever,

## PYELONEPHRITIS AND PYELITIS

rapid wasting, and profuse pyuria, no improvement is shown and the patient continues to go rapidly downhill, nephrectomy is not only justifiable but advisable. In others, if, after prolonged illness of some months with irregular periods of pyrexia interspersed with bursts of pyuria, the cystoscope shows the disease to be confined to one side, the affected kidney should be explored and a pyonephrosis drained or removed. In *bilateral* cases of a similar nature something can be done by daily lavage of the bladder, and, if renal suppuration still continue, ureteral catheters should be passed and an attempt made to improve the condition by washing out the infected pelvis and calyces with weak solutions of urinary antiseptics.

### 2 SECONDARY PYELONEPHRITIS (SURGICAL KIDNEY)

**Pathology.**—By secondary pyelonephritis is meant the infection of a kidney already embarrassed either by some local condition, such as stone, or by the effects of some long-standing obstruction lower down in the urinary tract, such as an enlarged prostate, a urethral stricture, or an hypertrophied and infected bladder containing a stone. In the latter group the renal pelvis and calyces show slight dilatation and thickening at the expense of an atrophied and sclerosed parenchyma. An ascending infection, which is almost always a bilateral one, is facilitated by the destruction of the valvular character of the lower orifice of the ureter, which has occurred as a result of the obstruction to the urinary outflow.

**Symptomatology.** (a) **Acute secondary pyelonephritis.**—The renal infection may occur spontaneously, but more often follows some surgical operation upon the lower urinary tract or the passage of instruments along the urethra. Within a few hours the temperature rises to 103–105° F., and the onset is marked by rigors and vomiting. The patient looks and feels very ill, and may complain of a certain amount of aching lumbar pain. The urine is foul and diminished in amount, or may be suppressed. The patient quickly passes into a condition of uræmia and becomes very drowsy. There is a dry furred tongue, much tympanitic abdominal distension with constipation, and some tenderness in one or both loins. The temperature may gradually fall as the pulse-rate rises and become subnormal, but usually is of the intermittent type, with sharp rises to 103° or 104° F. The pulse is small and

thready, and in the course of a few days the patient gradually sinks into a condition of coma and dies. Convulsions are uncommon. In some of the slighter cases the excretion of urine is gradually re-established and the patient partially recovers, only gradually to relapse into a chronic septic condition.

(b) **Chronic secondary pyelonephritis** is usually the sequel to a slight acute attack, but may come on insidiously in a case of chronic urinary obstruction. It is always bilateral. The patient has a sallow emaciated appearance, with a dry, glazed tongue and offensive breath. He is often very tremulous. The temperature may run a subnormal course for several weeks at a time. Mentally he is very dull and suffers from persistent frontal headache. He passes increased amounts of urine which are of low specific gravity, 1,005 to 1,008, and hazy with bacilluria and finely diffused pus. In this condition he may continue for a period of months or even years, but the change is a slowly progressive one and finally he becomes rapidly worse, little if any urine is passed, and the last phase ends fatally in two or three days from the date of onset of the acute symptoms.

**Treatment.** (a) **Acute secondary pyelonephritis.**—Every effort should be made to re-establish the excretion of urine by encouraging the patient to take large quantities of fluids, reinforcing this if necessary by the intravenous infusion of saline or glucose solution. Hot fomentations should be applied to the loins, and, provided that a careful watch be kept upon the pulse, a hot-air bath may be utilized. Calomel 5 gr., followed by a saline aperient, should be given, and the abdominal distension relieved by turpentine enemata.

Any obstruction to the lower urinary tract should be eliminated by a temporary suprapubic cystotomy and, if the patient survive, the actual lesion dealt with at a later date. If, in spite of these measures, suppression persists, one or both kidneys should be exposed from the loin, the outer convex border freely incised, and a rubber drainage tube inserted into the renal pelvis.

(b) **Chronic secondary pyelonephritis.**—If the renal condition has not progressed to a dangerous degree, an operation should be performed to remove the cause of the obstruction, after a preliminary period of bladder drainage through a suprapubic cystotomy wound. In this way the ultimate destruction of the kidneys may be delayed and in a few instances permanently arrested.

HAROLD W. WILSON.

## PYLEPHLEBITIS, SIMPLE

**PYLEPHLEBITIS, SIMPLE.**—A non-suppurative thrombosis of the portal vein, sometimes called *Pylephlebitis Adhæsiva*.

**Etiology.**—Infection of the wall of the vein is the cause in all cases, with the possible exception of those due to invasion of the lumen by new growth. In chronic cases there may be calcification of the wall of the vessel. In some cases no primary cause can be discovered, but the majority depend on disease of neighbouring organs, such as cirrhosis of the liver, malignant disease, tuberculous peritonitis, and diseases of the spleen or pancreas. Syphilitic endophlebitis sometimes produces it. It is commoner in males than in females, and the average age is 44.

**Pathology.**—The intima of the vein may be swollen and red, or thick and calcareous, and the clot itself may be firm and completely occlude it, or may be canalized. The liver may be unchanged or fatty, and sometimes contains infarcts. The spleen is usually enlarged and often infarcted.

**Symptomatology.**—The onset may be masked by the primary disease, especially when it is gradual. In other cases it is sudden and is ushered in by severe hæmatemesis and rapidly developing ascites. If the splenic vein becomes blocked the chief signs are profuse hæmatemesis and rapid enlargement of the spleen, and if the mesenteric vein is occluded, diarrhoea, melæna and symptoms of intestinal obstruction appear. Abdominal pain is nearly always present. In most cases which survive the early dangers, dilated abdominal veins become very prominent.

**Diagnosis.**—The disease is very difficult to diagnose. The chief distinction from *cirrhosis* is the rapidity with which the signs and symptoms develop, but a certain proportion of cases of cirrhosis exhibit similar features. *Splenic anæmia* resembles it in having splenic enlargement and hæmatemesis, but differs in its slow progress, in the presence of anæmia, and in the absence of dilated abdominal veins.

**Prognosis.**—If the patient survive the acute stage, life may be prolonged for many years, owing to the establishment of a collateral circulation, but recurrent hæmatemeses are usual and the general health is much impaired.

**Treatment.**—Ascites and hæmatemesis require the same treatment as in cirrhosis of the liver (*see LIVER, CIRRHOSIS OF*). The spread of the thrombus may be checked by potassium

## PYLEPHLEBITIS, SUPPURATIVE

citrate in 30-gr. doses, but this should not be given unless the coagulability of the blood has been examined and found to be high. It is already low in many cases of cirrhosis.

E. A. COCKAYNE.

**PYLEPHLEBITIS, SUPPURATIVE.**—A suppurative inflammation of the portal vein or its branches.

**Etiology.**—This disease, which is rare, is usually secondary to some suppurative lesion in the abdomen, but sometimes no primary source can be found. Appendicitis is the cause in about half the cases. It is commonest when there is a local abscess under pressure. Usually it develops early, but it may appear after appendicectomy. The other causes are very varied, and none is common. In infants the disease may follow umbilical infection.

**Pathology.**—A septic clot may fill the portal vein or one or more of its branches. The liver is much enlarged, and on section may appear to be honeycombed with small abscesses. This appearance is due to cross-section of the suppurating strands, which follow the course of the portal canals. Various organisms, including *B. coli*, streptococci, staphylococci, and pneumococci, have been found in the pus.

**Symptomatology.**—The onset is sometimes obscured by the primary disease, but is generally marked by sudden acute symptoms. Some are due to the pyæmia, such as pyrexia, intermittent, remittent, or continuous, rapid pulse and respiration, rigors, sweats, and leucocytosis. Others are ascribable to the local condition, which causes vomiting, diarrhoea, hepatic pain and tenderness with enlargement of the liver. Slight jaundice is present in half the cases. The patient looks very ill and, if the disease lasts long enough, becomes wasted and prostrated. When the abscesses are superficial, local peritonitis may lead to subphrenic abscess or empyema, or peritonitis may become general. Towards the end the temperature falls below normal and the patient becomes comatose.

**Diagnosis.**—This is rarely made. Pain, tenderness and enlargement of the liver, with fevers and rigors, in a patient known to have some intra-abdominal inflammation, are in favour of pylephlebitis, but are often regarded as signs of septicæmia and general peritonitis.

**Prognosis.**—Once established, the disease is always fatal.

**Treatment.**—If there exists great local

## PYLORUS, CONGENITAL HYPERTROPHIC STENOSIS OF

enlargement, suggesting the presence of a solitary abscess, exploratory laparotomy is justifiable; otherwise treatment must be palliative. Opium is the best drug for the relief of pain. Vaccine- and serum-therapy are disappointing.

E. A. COCKAYNE.

**PYLORUS, CARCINOMA OF** (*see* STOMACH, CANCER OF).

**PYLORUS, CONGENITAL HYPERTROPHIC STENOSIS OF.**—A congenital narrowing of the pylorus, due to antenatal hypertrophy of its circular coat.

**Etiology.**—Two contrary views have long been held regarding the causation of this condition: one that it is a true congenital malformation, the other, that the hypertrophy is produced by spasm and takes place within the first few weeks of life. There can, however, be little doubt that the hypertrophy precedes birth, for not only has it been detected in the foetus, but also the hypertrophy is too considerable to have taken place in the short interval which elapses before symptoms begin. On the other hand, there are difficulties in regarding it as a pure malformation. Were it a malformation it would be the only one of its kind, and, moreover, it is not accompanied by other congenital malformations. An alternative view is that the hypertrophy is produced during intra-uterine life by some agency which causes an hypertrophy of the circular muscle, for such an explanation meets the difficulty that it is congenital and yet not a malformation. Tyrrell Gray and Pirie ascribe it to antenatal spasm of the muscle produced by hyperadrenalism, but this hypothesis is, at present, based only on theoretical grounds. Moreover, there is no analogy for the supposition that pure spasm is capable of producing hypertrophy, though a muscle whose vigour of contraction is enhanced may certainly undergo this change. Although it may be conceded that the hypertrophy is congenital, some further explanation is needed to account for the obstruction. It is known that a considerable degree of hypertrophy may exist in the absence of symptoms of obstruction, that measures which do not remove the hypertrophy lead to the removal of symptoms and restitution of health, and that the symptoms rarely date from birth. The chief additional factor which determines the obstruction and consequently the onset of symptoms is probably pyloric spasm. Tyrrell Gray and Pirie hold that a

potent cause of this added spasm is phimosis. Evidence of obstruction in pyloric stenosis is much more common in male babies, and in them phimosis is a very common accompaniment. Relief of the phimosis is often followed by disappearance of the symptoms of pyloric obstruction. Moreover, phimosis may alone be responsible for obstruction in the absence of an hypertrophied pyloric muscle. Another possible cause of symptoms of obstruction is swelling of the gastric mucosa due to gastritis, set up by retention of food.

According to the authors named, with closure of the orifice a vicious circle is set up. The resulting absence of acid chyme in the first part of the duodenum leads to failure of secretin production and consequently to suppression of pancreatic secretion. The alkalinizing effect of the pancreatic secretion in the duodenum is therefore lost and the dilatation of the sphincter thereby inhibited.

**Morbid anatomy.**—The hypertrophy of the pyloric sphincter produces a tumour which is usually white and glistening, almost devoid of visible blood-vessels, and very dense in texture, being almost cartilaginous in its hardness. Its size varies from that of a hazel nut to that of an acorn. The smaller tumours are somewhat less hard and more vascular than the larger. Section shows that the swelling is almost entirely due to hypertrophy of the circular muscle, but the longitudinal muscle may also be hypertrophied to a less extent. The swelling ends abruptly at its duodenal end, but towards the stomach slopes more gently and blends with an hypertrophied gastric wall. This hypertrophy of the muscle of the stomach-wall may extend for several inches away from the pylorus, gradually diminishing in thickness until it is no longer detectable. The gastric mucosa is often swollen and injected, and covered with patches of mucus, changes which are most marked towards the pylorus. The pyloric orifice is seldom completely occluded, though narrowed by the constricting band of hypertrophied muscle. The stomach is often dilated to a greater or less extent, but this is by no means invariable. Apart from a phimosis, the other changes commonly met with after death are resultants of malnutrition and lowered resistance, and include general atrophy, enteritis, cystitis, pyelitis and pyelonephritis, and broncho-pneumonia.

The phimosis is caused by a tight prepuce stretched over a large glans, and the prepuce is not generally elongated.

## PYLORUS, CONGENITAL HYPERTROPHIC STENOSIS OF

**Symptomatology.**—Symptoms generally appear from a fortnight to three weeks after birth. Occasionally they date from birth, or they may be postponed for as long as four months. In one case I have seen they did not arise until the eighteenth week. When the infant is first brought for treatment there is a history of forcible, projectile vomiting, of constipation and of wasting. The vomiting may occur after every meal or may be only occasional, when not only is the last meal brought up but also the food which had accumulated since the last vomit. The vomited matter may be expelled across the cot and on to the floor beyond. It contains curds and mucus, but no bile. Occasionally it is bloodstained, and rarely there may be a brisk hæmatemesis. The stools are hard and infrequent. In appearance there is an almost uncanny similarity between infants who are suffering from pyloric stenosis. It is difficult to explain, but perhaps may be accounted for by the gradual wasting with retention of appetite and absence of pain, a combination of symptoms not usually seen in wasted infants.

Examination of the abdomen reveals the most important sign, i.e. a palpable pyloric tumour. It is usually found at the outer border of the right rectus muscle in the trans-pyloric plane, but may be dragged much farther down by a dilated stomach, may be far back against the vertebral column or tucked up under the liver. To the palpating fingers it seems a small hard ball, about the size of a hazel-nut or smaller, and it slips from under them. This tumour can almost invariably be detected if examination be made during or directly after a meal, and be repeated if unsuccessful on the first occasion. It is the only absolute sign of this disease. Next in importance is visible gastric peristalsis, which sweeps across from the left costal margin to the region of the tumour. It is often very considerable.

**Diagnosis.**—Intractable projectile vomiting with constipation and wasting, dating from the first two or three weeks of life, and accompanied by a pyloric tumour and visible peristalsis, constitute a clinical picture which can only belong to hypertrophic stenosis of the pylorus. In the absence of a palpable tumour the condition may be simulated by *pyloric spasm*, which is relatively rare. In its medical treatment or the relief of phimosis, if it exist, is followed by prompt amelioration, and a palpable tumour is never found. Radiograms after barium meals have been used extensively

as a diagnostic measure in America, but are unnecessary if a tumour is felt, and useless to distinguish stenosis from spasm when a tumour is not detected.

**Prognosis.**—Hitherto the mortality has been very high. In 54 cases admitted to the Hospital for Sick Children, Great Ormond Street, it was as much as 80·5 per cent. In private practice the mortality has been less. Sudden death sometimes occurs in an unexplained manner in patients whose obstruction is not severe, and whose general condition is good. The gradual enfeeblement from malnutrition is often brought quickly to an end by a fatal gastro-enteritis, which is a complication to be feared when suitable treatment permits a larger amount of food to reach the bowel. Other serious complications are broncho-pneumonia and infection of the urinary tract. The latter has been the cause of death in two cases recently under my observation.

Tyrrell Gray and Pirie have pointed out that the symptoms are slower in development and take longer to disappear in girls than in boys.

**Treatment.**—A choice between medical and operative treatment must be made. It is certain that many cases will end in complete recovery from symptoms on medical treatment alone. Others demand surgical procedures.

**Medical treatment.**—The infant should be kept quietly in its cot, and warmth ensured by hot-water bottles. Three or four small ones, which can be refilled in rotation, are better than one large one. If the infant is breast-fed, this method of feeding should be adhered to. I have found that five-minute feeds at the breast every hour, so that only the poorer milk is used, sometimes leads to prompt diminution of the vomiting and rapid general improvement. Under this régime the breast should be emptied by a breast-pump after every third feed. When breast milk is not available, only small quantities of an easily digestible artificial food can be given. The most satisfactory is peptonized milk, which may be given in the usual quantities for the child's weight, and need not be given more frequently than customary—certainly not more often than ten times in the twenty-four hours. If vomiting is considerable, a beginning may be made with peptonized skimmed milk.

The stomach should be washed out regularly through an œsophageal tube, with a mildly alkaline solution (1 dr. of sod. bicarb. to  $\frac{1}{2}$  pint of warm water). This should be repeated either once or twice a day, according to the

## PYLORUS, STENOSIS OF

amount of curd and mucus removed. Drugs are of little avail. The constipation is merely an indication of the small amount of food getting into the bowel, and purgatives are contraindicated. If difficulty in passing the dry motions is encountered, it is best combated by rectal lavage or by a small oil enema. If phimosis exists, it should be remedied at the beginning of treatment with a view to removing a possible cause of pyloric spasm.

Under such a course of treatment improvement must not be expected immediately, even in favourable cases, and a rapid improvement in weight is to be deplored and often precedes a serious mishap. In a considerable number, however, steady and persistent improvement begins in about ten or twelve days.

**Surgical treatment** is indicated in the cases of severe stenosis in which symptoms date from birth, for in these the organic obstruction is itself sufficient to produce symptoms, and spasm plays but a small part. It cannot be delayed, too, in very puny, wasted infants; in addition to the regimen described above, these should receive, hypodermically, saline infusions containing 2 per cent. of glucose. If improvement, as gauged by the vomiting, size of stools, and weight, does not follow within two or three days, surgical treatment should be resorted to. In the remaining less severe cases, where symptoms have not dated from birth, and where the infant's condition is less serious, it is more difficult to decide when an operation is called for. In the absence of improvement it should not be postponed longer than twelve or fourteen days, while serious loss in weight or intractable vomiting may demand its employment sooner. A somewhat longer period may be permitted in the case of girls.

The operations which have usually been employed are Loreta's operation, gastro-enterostomy, and that devised by Rammstedt, which consists merely in splitting the pyloric muscle by a longitudinal incision along its anterior wall and leaving the pyloric tube of mucous membrane to fill the gap so formed. The last is the operation of choice, for it is rapid, precise, and practically devoid of shock. A postoperative rise of temperature to 104° F. is to be expected, but seems to have little effect on the general condition. An ounce of peptonized milk may be given four hours after the operation, and thereafter every four hours for the first twenty-four hours. The milk may then be given every three hours, 1½ oz. at a time, for another twenty-four hours, and

## PYONEPHROSIS

subsequently gradually increased. The peptonization may generally be abandoned after ten to twelve days, and a gradual return made to normal quantities and frequency.

FREDERICK LANGMEAD.

**PYLORUS, SPASM OF** (*see* STOMACH, FUNCTIONAL DISORDERS OF).

**PYONEPHROSIS.**—A lobulated swelling of globular or reniform shape, derived from a kidney the secreting substance of which has been partially or wholly destroyed by the combined processes of retention and suppuration. The kidney may show little or no enlargement, but in extreme cases may form a tumour occupying the greater part of the abdominal cavity.

**Pathology.**—A pyonephrosis consists of a number of imperfectly separated compartments, continuous with the dilated renal pelvis and calyces, and filled with pus. The walls of these loculi are formed by the tissues of the renal parenchyma, which may show various degrees of degeneration, or may be represented merely by a firm fibrous mass. The tumour is surrounded by an adherent fibro-lipomatous sheath, derived from the fatty capsule of the kidney, and may contain numerous secondary phosphatic calculi.

**Varieties.** (A) **Cases of hydronephrosis which have become secondarily infected.**—The commonest infecting agents are *B. coli*, staphylococci, and *B. proteus*. The hydronephrosis may be either unilateral or bilateral.

i. **Unilateral.**—The most frequent causes of this condition are a stone impacted in the renal pelvis or ureter, pregnancy, stricture of the uretero-pelvic junction, and movable kidney associated with an aberrant renal artery. The greater part of the tumour is formed by the greatly ballooned and thickened pelvis, while the sclerosed kidney, dilated to perhaps twice its natural size, is superimposed upon one aspect of the swelling. The organisms reach the kidney either by the blood-stream or by direct invasion from the bowel.

ii. **Bilateral.**—Here the cause of the original condition is some obstruction in the lower urinary tract, such as enlargement of the prostate or urethral stricture. In ordinary circumstances an intact ureteric opening is an effective barrier to an ascending infection, but when its valvular arrangement has been interfered with and the dilated ureter is in free and open communication with the bladder,

## PYONEPHROSIS

organisms can readily pass upwards in the resulting stagnant column of urine and thus reach the pelvis of the kidney.

(B) **Microbic infection of apparently healthy kidneys (acute primary suppurative pyelonephritis).**—In the great majority of these cases the condition is unilateral, occurring in women and children, and due to bacilli of the coliform group. The renal pelvis is little, if at all, dilated, and the tumour is entirely composed of the enlarged and dilated kidney. (See also PYELONEPHRITIS AND PYELITIS.)

**Symptomatology.**—The cardinal symptoms are fever, pain and pyuria, in association with a tumour in the loin.

1. **Fever.**—The temperature almost invariably rises slightly at night to 99·5° or 100° F., and as a result of septic absorption the patient is usually pale and thin. At intervals of a few days or weeks, however, when blocking of some portion of the ureter coincides with an absence of pus in the urine, the temperature may rise to 102° F. or even higher. An intermittent fever ranging widely up to 103° or 105° F. often denotes perinephric suppuration.

2. **Pain.**—An almost constant lumbar ache, worse at night, is sometimes associated with mild attacks of ureteral colic.

3. **Pyuria.**—The urine usually contains much pus, which settles down as a heavy mass at the bottom of the specimen glass, the supernatant liquid having a hazy or milky appearance. A temporary absence of pus from the urine may be associated with an accession of fever, and an increase in size and tenderness of the tumour.

4. **Tumour.**—The well-defined globular or reniform swelling is situated deeply in one or other loin, usually in the right. It feels tense and hard. If it occurs on the left side, the descending colon can often be seen and felt on its anterior surface. Some tenderness and muscular rigidity may be detectable in the loin of the affected side. The mobility of the tumour varies. In acute primary pyelonephritis, it moves on respiration and a certain amount of lateral mobility can be conveyed to it by the examining fingers; on the other hand, a calculous pyonephrosis of some years' standing is absolutely fixed.

**Diagnosis.**—The diagnosis is made on the history of the condition of the urine, the characters of the abdominal swelling, and the fever associated with pyuria. Cystoscopic examination will often reveal continuous columns of pus being slowly extruded from the ureteric opening of the diseased side, and ureteric

## PYORRHŒA ALVEOLARIS

catheterization may show that the opposite kidney is unaffected.

**Treatment.**—The best treatment for unilateral pyonephrosis is nephrectomy by the lumbar route, the tumour, together with the diseased portion of the ureter, being removed from within its capsule of thickened and adherent perinephric fat. If the patient be desperately ill, a drainage operation may be all that it is possible to attempt at first, but it is invariably followed by a persistent sinus. In these circumstances secondary nephrectomy must be considered at a later date. At this stage, when profuse perinephric suppuration has been in existence for a period of several weeks or months, it is a difficult and dangerous procedure. In bilateral cases, both tumours should be drained from the loins, if the patient's general condition be such as to warrant surgical interference.

HAROLD W. WILSON.

**PYOPERICARDITIS** (see PERICARDITIS).

**PYOPNEUMOTHORAX** (see PNEUMOTHORAX).

**PYORRHŒA ALVEOLARIS.**—A state of chronic infection of the tissues surrounding the teeth. The name implies a discharge of pus from a tooth socket, but it is only under certain conditions of secondary infection that pus can be seen.

Many micro-organisms have been described, but none can be regarded as specific. An acute form, known as "trench gums," met with so frequently during the War, was found to be due to the organisms of Vincent's angina. Pyorrhœa alveolaris must be regarded as a chronic disease which becomes more common as age advances. It usually appears after the age of 30, and is essentially an affection of adults. Those who persistently keep their lips apart — mouth-breathers — almost invariably suffer from this condition, and in these patients the stage of gingivitis commonly occurs during childhood, but rarely passes to a more advanced state before the age of about 20. Several varieties of the affection may be distinguished as knowledge advances, but at present it is not possible to do more than indicate variations in its course.

Infection occurs at the gum surface against the tooth, involving the periodontal membrane and then the bone of the socket. The bone infection constitutes the serious feature of the disease, and, if this be extensive, treatment by retention of the teeth will be unsatisfactory.

## PYORRHEA ALVEOLARIS

**Clinical course.**—Swelling appears in the gums, beginning in the papilla of gum between the teeth; it is congested, bluish, soft, bleeds easily, and is found to be detached from the tooth, so that a minute "pocket" is produced, even though the destruction be but slight. Food particles lodge readily and undergo decomposition, adding to the local irritation; the periodontal membrane is involved, and in due course the bone. The apex of that portion of the alveolar process situated between the teeth undergoes absorption, and as this progresses a space appears, in spite of the swelling of the gum papilla. The first indication of bone destruction is a small triangle of congested gum papilla between two teeth, the base of the triangle being a definite line passing between the necks of the teeth, produced by gum folding over the edge of the bone remaining. Later, all the tissues surrounding the teeth are involved, and the gums become swollen over the corresponding alveolus, but chiefly its marginal portion. At this stage of gingivitis and slight bone destruction the disease can be treated satisfactorily, but it is more serious when the bone becomes further diseased. The infection may extend into the bone without causing obvious destruction, or the absorption may be evident to the naked eye. When developed, the disease affects the gums, the periodontal membrane, and the alveolar processes of the jaws. The tissues beyond those around the tooth are so rarely invaded that the infection would appear to travel along the periodontal membrane primarily. Local examination, with a study of radiograms, suggests that this may not always be true, but destruction of the compact layer of bone forming the socket occurs in all cases in which the infection is extensive.

Rarefaction always occurs if the progress is rapid or prolonged (although in the latter event sclerosis may occur concomitantly), the teeth become loose, and in due course may "drop out." Before this happens, the movement of the tooth usually leads to the formation of strong bands of fibrous tissue, binding it to the alveolus, so that, in spite of its looseness, difficulty is often experienced during extraction. The difficulty may be added to by the tooth having become more brittle, an almost constant change in these cases. The sclerosed bone is peculiarly dense and inelastic.

The destruction of bone usually proceeds from the neck of the tooth towards the apex; on one aspect of the tooth the bone may be

completely destroyed, while remaining more or less intact elsewhere. Occasionally a fine track along the side of the tooth may extend as far as the apex, leading to infection of the pulp or, if drainage be imperfect, to an abscess involving the bone of the socket. Symptoms are unusual, except in the later stages, probably on account of the drainage which occurs along the side of the tooth.

The disease may continue for years and the gum tissue undergo absorption, particularly between the teeth, so that bare bone can often be felt; the changes are such that a sharp dental probe can be forced into it without causing pain, but the periodontal membrane is still sensitive. If the destruction is uniform, radiograms show the bone intact around the deeper aspect of the socket; if it is unequal, it is seen to have spread more irregularly, involving one part of the socket chiefly. Cases of the former group are far more favourable for treatment by retention of the teeth. The two changes may be present in the same mouth, or may exist apart. Pus may be squeezed from a pocket, but food particles and débris are constantly found and frequently mistaken for pus.

A form is met with in young adults from 20 to 30 or even younger, in which much destruction of tissue is seen; the gums are not greatly swollen, but become smooth and have a bluish appearance; it is common to find pus, and the saliva is very plentiful. The destruction of bone is rapid, and occurs immediately around the tooth; sclerosis is rare. An appearance of swelling of the gum is produced by its folding over the bony edge of the outer part of the socket which remains, that near the tooth having been lost. Teeth affected to any degree require extraction, for infection from one tooth to another is almost certain. Mouth-breathing is frequently present.

In another form there is considerable sclerosis of the bone with overgrowth beneath the alveolar periosteum. The irregular nodules which are readily felt over the roots of the teeth are characteristic, and though found in all forms, they are much more definite in this. The margin of the socket is often destroyed, and as bony thickening has occurred beyond, the gum folding over it has a square-cut appearance and looks more swollen than it actually is. The gingival tissues may be thickened by a local fibrosis. At times enormous thickening is seen, leading to hyper-



## QUARANTINE

trophy so pronounced that the condition is described as a separate disease. The hypertrophy is very largely of bone, and is greatest in the molar region, especially of the maxillæ.

**Symptoms.**—Because of its slow progress, local symptoms are not prominent. General symptoms, however, are not uncommon, and may be the first indication that the teeth are affected (*see* ORAL SEPSIS). Patients who exercise great care with their teeth may render them so clean superficially that for a long time the disease may not be detected.

An unpleasant taste in the mouth after sleep is an early symptom, but does not usually occur until the condition is so far advanced that extraction of at least one tooth is the only safe line of treatment. Bad dental restoration must always be kept in mind in such cases.

Local secondary infections may be present, such as sore throat, ulceration of the mucous membrane of the mouth, impetigo of the skin of the lips and cheeks, soreness of the tongue, ulcers following slight injury—for instance, from a toothbrush—and those so commonly found at the bottom of the sulcus between the gums and cheeks and lip, particularly of the lower jaw. Of considerable interest is a soreness and redness of the tongue in the region of the papillæ foliata, which seldom amounts to ulceration.

Tenderness of the teeth may be noticed when they are bitten upon, and a desire to grind or press the teeth together is common. The destruction of tissue exposes the root of the tooth, which is very sensitive, and the pain of caries may be so closely imitated that patients often cannot believe that a cavity does not exist. A focus so small that it is difficult to find may cause great discomfort. Exact changes in the roots of the teeth *in situ* can be recognized only by means of radiograms, but without this aid an examination of the mouth often suffices to give a correct impression.

**Treatment** can be carried out satisfactorily only by a dentist. That described under ORAL SEPSIS should be adopted prior to referring the patient to a dentist. Vaccine treatment has been practised extensively, but with little success for the local condition until all local irritation has been removed and favourable progress made. It gives the best results in some cases of secondary infection, but only after the mouth has been rendered healthy.

WARWICK JAMES.

**PYO-SALPINX** (*see* SALPINGO-OÖPHORITIS).

**PYROSIS** (*see* STOMACH, FUNCTIONAL DISORDERS OF, p. 269).

**PYURIA** (*see* URINE, EXAMINATION OF).

**QUARANTINE.**—When an infectious disease breaks out in an institution such as a hospital, school, or barracks, all who have been exposed to infection, unless protected by a previous attack, are liable to contract and disseminate the disease. It is usual, therefore, to place such "contacts" in quarantine, i.e. to isolate them for a certain period from all susceptible persons who have not been exposed to infection. By this means, if the precaution has been taken in time, a spread of the disease is in great measure prevented.

During the quarantine period, careful supervision is required to prevent fresh exposure to infection, and disinfection of the person and clothing should be carried out. Children in whose families infectious disease has recently

occurred should not be allowed to attend school, and an infected hospital ward should have no fresh cases admitted to it.

The period of quarantine, which, as the name implies, was originally 40 days, varies with each disease. It corresponds with the incubation period of the disease in question, two or more days being added as an additional safeguard.

The following table shows the quarantine period for the commoner infectious diseases:—

Scarlet fever . . .	7 days.
Mcasles . . .	21 "
Rubella . . .	21 "
Smallpox . . .	21 "
Chickenpox . . .	21 "
Whooping-cough . . .	21 "
Mumps . . .	26 "

## QUINSY

Diphtheria "contacts" may be released from quarantine when the cultures of the nose and throat on two successive days have proved negative.

By the Public Health Act of 1896, which repealed the old Quarantine Act, the port sanitary authority is authorized to detain and examine a vessel arriving with any person on board suffering from a dangerous infectious disease, especially cholera, yellow fever, or plague. The case is removed to hospital, the names and addresses of the healthy passengers are taken for the information of the medical officer of health at their destination, and the vessel with its cargo is disinfected. Suspected cases may be detained for a period not exceeding two days.

J. D. ROLLESTON.

**QUINCKE'S DISEASE** (see Angio-neurotic Oedema, under **ŒDEMA**).

**QUINSY.**—An abscess occurring in the peritonsillar tissue.

**Etiology.**—Quinsy is commoner in males than females, and usually occurs between the ages of 14 and 40. *Predisposing* causes are all conditions which tend to lower vitality, such as bad hygienic surroundings and poor food. The condition is also predisposed to by septic conditions in the mouth, nose, nasal accessory cavities or ears, and enlarged tonsils. The *exciting* cause is infection with a micro-organism, the most frequent being the *Streptococcus pyogenes*.

**Pathology.**—The infection commences frequently as an acute tonsillitis, and passes on to attack the peritonsillar structures. Often, however, it commences in the latter region. The palate and uvula become inflamed and œdematous, and pus forms in the course of a few days, leading to the production of an abscess occupying the peritonsillar tissues.

**Symptomatology.** The leading symptoms are pain, inability to swallow, altered voice, which becomes thick and dead, excessive salivation, inability properly to open the mouth, and rigidity of the muscles of the neck. These symptoms are initiated usually by a chill with a rise of temperature. On examination a unilateral inflammation of the palate, with œdema of the uvula, is usually seen; the tonsil on the affected side is found to be pushed inwards and to be encroaching on the lumen of the

pharynx; the tongue is coated and foul, and there is free salivation. The general condition of the patient is poor.

**Prognosis.**—The condition usually lasts from four to twelve days. Once peritonsillar inflammation sets in, it rarely clears up without abscess-formation. Relief occurs with the rupture and evacuation of the abscess.

**Diagnosis.**—This presents no difficulty, as the symptoms are definite, and examination shows the typical appearance.

**Treatment.**—All attempts to abort abscess-formation result in failure. In the first instance, put the patient to bed and evacuate the bowels with 2-5 gr. of calomel at night, followed by a morning saline. The mouth is best kept clean with a spray of hydrogen peroxide and a mouth-wash of sanitas—one teaspoonful to half a tumblerful of water. Internally, a mixture containing 10 gr. of salicylate of soda, with 3 gr. of phenacetin and 2 gr. of citrate of caffeine, taken every two hours for four to six doses, gives relief to the pain. Sulphate of quinine in 5-gr. doses thrice daily is useful.

When the abscess has formed, the only relief is by opening and evacuating its contents. Picturesque details given in textbooks as to the best point to open will only lead the practitioner astray. The point at which to open is the most bulging point; this is almost invariably above the outer side of the tonsil. The common mistake is to open too close to the anterior pillar, and consequently into the tonsillar tissue instead of into the distended peritonsillar cavity. The routine of opening is as follows: First, cocaineize the inflamed bulging area with a spray of 10-20 per cent. cocaine, carefully instructing the patient to spit this out promptly: this can be done twice or thrice at a few minutes' interval. Then with a sharp-pointed tenotomy knife make an incision not more than a quarter of an inch deep into the most prominent part of the bulge. If this does not strike the pus, follow up by pushing in a sinus forceps; this will usually open the abscess cavity. In withdrawing the forceps, open them well out, so as to get a good opening for drainage. The subsequent treatment consists in having the mouth and pharynx kept clean with the hydrogen-peroxide spray and sanitas mouth-wash, and in administering iron and strychnine internally.

J. GAY FRENCH.

## RADIANT HEAT, TREATMENT BY

**RABIES** (*see* HYDROPHOBIA).

**RACHITIS** (*see* RICKETS).

### RADIANT HEAT, TREATMENT BY.

—By this is usually meant treatment by the use of some form of incandescent carbon-filament electric-light cabinet. This is chiefly a thermal bath, for the luminous efficiency is only about 5 per cent. of the total output. Still, when some 60 or more lamps are used, as in a general light bath, the luminous output assumes considerable dimensions, and in this are found luminous frequencies from violet down to the red, so that there is a considerable chemical efficiency in the incandescent-light bath. The effects of such a bath, however, must not be confounded with the effects of the electric-arc bath, which is essentially a chemical-light bath.

The heat reaches the tissues by radiation, not convection, and penetrates to a considerable depth, acting on all the tissues as well as the fluids of the body with which it comes into contact, carrying into them heat which is not rapidly dispersed by convection and conduction, and raising their temperature to a higher degree than with the convective methods of administration. The effect is not merely local but general. The skin is a poor conductor of heat, but transmits radiant energy readily. The heated blood-stream causes a warm, glowing sensation, and, if prolonged, awakens active secretion of the sweat-glands, the cooling influence of absorption by the latent heat of evaporation maintaining the body-temperature at normal. The penetrating luminous rays act upon the blood. Under long exposure the irradiated tissues become hyperæmic, the blood that passes through them being exposed all the time to the oxidizing and sterilizing influence of the light. The effect is general, and beneficial in conditions of impaired and perverted nutrition and poor metabolism. The general bath has a considerable influence on the heart, pulse, and tonicity of the arteries. I have found a pulse of 70 gain 12 beats per minute after ten minutes, and 34 beats after fifteen minutes, reaching 130 after twenty minutes. At the same time the respiration is quickened and becomes more shallow. I always give a warm bath after the general light bath, gradually cooling down by running in cold water. In this bath sinusoidal current may be admin-

istered if desirable. The patient is then lightly dried and covered with towels and dressing-gown, and allowed to lie on a couch till well cooled down, when he should dress deliberately and avoid any hurry or exertion, as it is easy to restart the activity of the sweat-glands and, perspiring again, the patient is very apt to catch cold.

This treatment I have found useful in cases of muscular soreness, lumbago, stiff-neck, and other myalgias arising from defective metabolism, exposure to cold or wetting of the clothing. I have also used it in cases of arthritis due to the influence of toxic absorption from the alimentary canal or elsewhere, so-called "rheumatism," and also in gonorrhœal rheumatism. The results, however, are by no means constant. The treatment seems to suit some cases and to make others worse. It is useful in obesity, biliary lithiasis, and diabetes; in some cases of altered blood-state, such as anæmia, chlorosis, lymphostasis, and syphilis; in diseases of the kidneys, such as nephritis; in respiratory and circulatory conditions, such as bronchitis, bronchial asthma, and cardiac hypertrophy. Cases of rheumatoid arthritis certainly show much greater improvement when the radiant-heat bath is followed by sinusoidal current in the warm bath, or by high-frequency effluvia subsequently administered. In cases of cardiac hypertrophy it is best to start with a fairly low temperature, gradually increasing the heat by switching on groups of lamps till a moderate temperature is obtained, but avoiding high temperatures altogether. The application should be brief in these cases, only lasting till gentle perspiration is induced. In cases of nephritis great care must be taken to avoid chill. A slight exposure of the body to the influence of evaporation might be sufficient to cause contraction of the blood-vessels and counteract the beneficial effects of the bath. After the bath the patient should immediately be wrapped in flannels and placed in bed. The use of the light bath is contraindicated in uræmic cases, organic heart disease accompanied by marked congestive symptoms, in phthisis with night sweats, in hæmoptysis, hæmatemesis, and apoplexy. For some years I have made great use of this mode of treatment in the Electrical Department of University College Hospital, but have gradually replaced it by other methods

## RADIUM-THERAPY

of treatment which appeared to me to be more desirable, until now the use of the general light bath is scarcely ever resorted to.

E. S. WORRELL.

**RADIOGRAPHY** (*see* X-RAYS, DIAGNOSTIC USES OF).

**RADIUM-THERAPY.**—The therapeutic properties of radium are dependent upon the effects of the rays which are constantly emitted by radium salts or radium emanation. These rays have been classified by physicists into three main groups— $\alpha$ ,  $\beta$ , and  $\gamma$  rays.

**Alpha rays.**—These rays are particulate, and composed of exceedingly small particles resembling the hydrogen atom in size. The particles lose their velocity and their activity in passing through a few centimetres of air. They are very easily absorbed, and have but feeble penetrative power, a layer of varnish, a sheet of paper or of the thinnest aluminium foil, sufficing to intercept them. They carry a positive electrical charge.

**Beta rays.**—While resembling cathode rays, these rays possess a much higher power of penetration. They consist of negatively charged bodies projected with velocities of the same order as the velocity of light. They are further subdivided according to their velocity into "soft," "medium," and "hard." The soft and medium beta rays are stopped by lead between 0.5 and 1 cm. in thickness, but some of the hard rays are able to penetrate even 5 cm. of that substance.

**Gamma rays.**—These are an undulation of the ether, and are analogous to Röntgen rays. They possess a high power of penetration and can pass through a thickness of 10 cm. of lead. They are not electrically charged.

**Secondary rays.**—The passage of gamma rays through screens of heavy metal is accompanied by the production of secondary rays, which are capable of producing severe surface irritation if allowed to act upon the skin, and they therefore have to be absorbed by the interposition of paper, lint, or rubber between the metal screen and the skin or mucous membranes. It has recently been shown that the emergent "secondary rays" from a leaden screen 2 mm. in thickness can be completely absorbed for all practical purposes by a layer of aluminium 0.2 mm. in thickness, and such leaden screens are now often made with this additional aluminium sheathing, which does

away with the necessity for the use of paper and lint.

Radium is employed in the treatment of disease either in the form of—

(A) **Radium salts**, which may be—

- (1) Mounted on varnished applicators;
- (2) Enclosed in small glass tubes, closely packed to prevent movement of the salt;
- (3) Dissolved in solution;

or (B) **Radium emanation**, which may be—

- (1) Enclosed in hollow flat metal applicators;
- (2) Contained in sealed capillary glass tubes;
- (3) Dissolved in solution.

For convenience of description, in speaking of the activity of varnished applicators a unit of concentration has been adopted. This consists of a centigram of radium bromide spread over a square centimetre. Applicators containing radium salts to this extent of concentration are said to be "full strength" applicators; similarly, "half" and "quarter" strength applicators contain respectively 0.5 and 0.25 cg. of radium bromide respectively to each square centimetre.

Radium emanation is used exactly as are the radium salts, due regard, however, being paid to its loss of activity by decay. The rate of decay follows a definite curve, the initial activity falling to half value in 3.85 days, to one-fourth in 7.6 days, and being practically inert at the end of a month, the emanation becoming transformed into helium.

**Screens.**—The screens used to filter off the different rays emitted from the radium apparatus are—

- (a) Aluminium: 0.01, 0.02, 0.04, and 0.08 mm. thick.
- (b) Silver: 0.5 and 1.0 mm. thick.
- (c) Lead: 0.1, 0.2, 0.4, 0.5, 1.0, 1.5, and 2.0 mm. thick.
- (d) Platinum: 0.3 and 0.5 mm. thick.

The aluminium and the thinner lead screens are used when it is desired to cut out only the  $\alpha$  and soft  $\beta$  rays; the silver, platinum, and thick lead screens when a radiation containing only the hard  $\beta$  and  $\gamma$  rays is required.

Special shapes of screens are often called for, particularly when it is desired to vary the character of the radiation that different portions of the same lesion shall receive.

## RADIUM-THERAPY

The most useful of these are:—

- (1) "*Window*" screens.—In the manufacture of these a leaden tube with walls 2 mm. in thickness is taken, and a window occupying one-third of its circumference cut in it. This gap is then closed with a screen of 0.1 mm. of lead, 0.5, or 1.0 mm. of silver, according to the nature of the surface treated. A tube of radium salt or radium emanation is then inserted into the tube, and the apparatus placed in the position which will permit the ulcerated or fungating portions of the growth to receive the lightly filtered radiation, and the remainder the heavily filtered rays.
- (2) "*Sleeve*" screens.—In these a silver tube, with walls of 0.5 or 1.0 mm. in thickness, is inserted in a collar or sleeve of lead 1.5 or 2.0 mm. in thickness, encircling half or two-thirds of the length of the silver tube.
- (3) "*Hour-glass*" screens.—Specially useful in the treatment of annular carcinomatous strictures of the rectum or œsophagus. Their shape ensures their retention in an accurate position throughout the exposure.

The duration of exposures varies from 2 or 3 minutes to 100 or more hours, according to the nature of the lesion treated and the strength of the apparatus and character of screening employed. Exposures of less than 2 hours' duration are usually given without a screen—except for a thin layer of rubber applied over the apparatus to protect the varnish. Such exposures are used in the treatment of superficial skin diseases, warts, keratomata, small rodent ulcers, etc. In epitheliomata, large excavated rodent ulcers and cheloid, exposures of 3–12 or 20 hours may be necessary, and screens of 0.1 mm. or 0.2 mm. of lead, or 0.5 and 0.1 of silver, should be employed.

In the radiation of deep-seated growths the use of the penetrating hard beta and gamma rays is requisite, and screening of 1.5 mm. or 2 mm. of lead must be resorted to, and a total exposure of 24–60 hours' duration given, spaced over a week or ten days.

When applying an apparatus, the surface of the lesion to be treated should be cleaned and dried, all crusts and secretions being removed. The surrounding healthy tissues should be protected with lead rubber sheeting, similar to that used in X-ray work, and the applicator kept in position with some non-irritant adhesive rubber plaster.

The employment of a tampon or light gauze

packing is necessary for the retention of tube apparatus in the vagina or uterus.

In the rectum, bladder, nose, mouth, and œsophagus the apparatus is attached to a handle of thick pliable silver wire which can easily be bent, and fastened in an appropriate position with strapping and a bandage.

**The reaction.**—All tissues when treated with radium respond in some manner, but the degree of this response varies greatly and is dependent upon the length of exposure, strength of apparatus, and class of screening employed. Other factors which play an important part in the reaction are the nature and condition of the tissues treated, and the size of the area exposed to the rays. Tissues of a definitely pathological character—carcinomatous or sarcomatous—do not display the same resistance to radium rays as normal cells, and it is upon the proper and correct appreciation of this fact that successful radium-therapy largely depends.

The reaction usually appears between the seventh and fifteenth days, but personal idiosyncrasy is occasionally responsible for curiously puzzling results, and with some patients the reaction will appear on the third or fourth day, with others it may be delayed for four or five weeks. Four degrees of reaction may be clearly distinguished: (1) Simple erythema; (2) erythema followed by desquamation; (3) vesication with simple ulceration; (4) deep ulceration of a destructive character, sometimes accompanied by the production of an eschar.

**Treatment of disease. Squamous-celled carcinoma (epithelioma).**—Surgical measures are always to be preferred to radium treatment in those cases where a complete and wide excision of the growth is practicable; but if operation be impossible or the patient refuse to submit to it, radium will often prove of great service, and in some instances, if glabrous surfaces only be affected, will bring about an apparent cure. Heavy dosage must, however, be resorted to, and the resultant reaction carefully observed, as an irradiation of insufficient intensity may act as a powerful stimulus to the lesion.

Epitheliomata of the face, trunk, or extremities, if flat and accompanied by little or no ulceration, give satisfactory results when treated with quarter- or half-strength apparatus, screened with 0.1 mm. of lead, the exposures varying from 6 to 12 hours' duration, spread over a period of three or four days. The retro-

## RADIUM-THERAPY

gression of the growth is quickly brought about, and little or no scarring results.

Ulcerating epitheliomata without much sub-jacent infiltration require treatment with quarter- or half-strength apparatus applied unscreened for a total exposure of 2 to 6 or 8 hours, spread over a period of four or five days, and repeated after an interval of six weeks if necessary. A destructive reaction follows, but the result is usually quite good, and there is but little cicatricial contraction.

Ulcerating epitheliomata with great infiltration require prolonged treatment with heavily-screened apparatus emitting only hard beta and gamma—the so-called “ultra-penetrating”—rays. Exposures of 30–60 hours given during one week, and repeated in five or six weeks’ time, are best; but if the growth show signs of rapid extension the borders should be given a vigorous treatment with unscreened apparatus.

Epitheliomata of the buccal, lingual, and pharyngeal mucous membranes usually prove both refractory and disappointing in their response to radium, but a method of treatment has been devised which, in instances of cancer of the tongue, has given somewhat encouraging results; it consists in the burying within the carcinomatous nodule of a very small but intensely powerful radium emanation tube, possessing an initial activity equivalent to that of 40 to 100 mg. of radium bromide, enveloped in a screen of 1 mm. of silver or 0.3 mm. of platinum, and giving an exposure of 6–24 hours’ duration. A fairly severe reaction follows, and in some cases the nodule ceases to grow and becomes replaced by dense fibrous tissue.

In epithelioma of the œsophagus, distinct though usually temporary benefit often follows on the introduction of a 100-mg. tube screened with 1 mm. of silver actually within the lumen of the growth, an exposure of 18–24 hours being given. That the effect is not merely a mechanical one due to dilatation of the stricture is shown by the fact that improvement in the power of swallowing may remain for six, nine, or more months after the termination of the treatment.

**Carcinoma of the uterus.**—This condition usually responds in a most gratifying fashion to radium treatment, and the results produced in inoperable cases are far in advance of those obtained by any other known medical or surgical methods.

The local manifestations of the disease are benefited in the most striking manner, and the

complete disappearance of fungating growth, arrest of hæmorrhage and discharge, healing of ulceration and relief from pain are phenomena of frequent occurrence. In favourable cases, moreover, the treatment appears to exert a distinctly retarding influence on the dissemination of deposits, and thus to arrest the progress of the disease. It is not possible, however, to speak of “cure” even in these latter instances.

When the uterus is very firmly fixed, the patient complaining of sacral and pelvic pain, radiating into both thighs, infection of the pelvic glands and deep parametric tissues may be regarded as certain, and radium is able to do but little for these patients, beyond possibly arresting the disease, and slightly diminishing the pain.

When treating cases of cervical carcinoma, if the growth be of the cauliflower or fungating type, the removal of as much of the mass as possible, by excision or curetting before the treatment is commenced, is to be advocated. A tube containing not less than 100 mg. of radium should be employed, the screening should be either 1 mm. of silver or 2 mm. of lead, according to whether the apparatus can be inserted actually within the cervical canal, or has to be placed in the vagina in contact with the diseased surface.

The uterine tissues appear to have a greater power of resistance to the radium rays than have those of the vagina, and the employment of a silver screen in contact with the vaginal wall for a period of more than 18 hours might quite possibly lead to the formation of a fistula, but with the tube inserted within the cervical canal this sequela need not be apprehended. The exposure should be one of between 20 and 24 hours’ duration, and may advantageously be supplemented by the use of a plate containing 100 or more mg. of radium screened with 2 mm. of lead, and applied externally over the fundus. By this means a thorough radiation of the whole diseased area is obtained. The exposures should be repeated at intervals of not less than six weeks.

Circumscribed and indurated nodules of small size in the substance of the cervix may receive, in addition, treatment by the insertion into their substance of tiny emanation tubes of 20 to 30 mg. initial activity, screened with 0.3 mm. of platinum, for from 12 to 18 hours.

In all cases in which radium has been applied within the vagina, the patient should be

## RADIUM-THERAPY

instructed to douche freely night and morning for at least six weeks after the termination of the treatment, as unless this be done an adhesive vaginitis not infrequently occurs. In some extremely susceptible subjects a transient proctitis has been noted to follow upon intravaginal treatment.

**Carcinoma of the breast.**—Radium treatment should never be resorted to as a substitute for surgical interference in operable cases of this disease, but when operation is impossible, or the patient has refused to submit to it, the employment of radium is indicated.

Many varieties of this condition occur, from the rapidly growing encephaloid type with extensive lymphatic involvement, to the localized hard atrophic tumour which may have existed for two or three years without occasioning the patient any pain or discomfort.

The prognosis is invariably bad in cases of the former character, more especially when the disease occurs, as it so frequently does, in young women of corpulent habit; and but little real benefit is to be expected from the use of radium, though prolonged screened exposures to the periphery of the growth may occasionally retard the progress of the disease to some slight extent.

The atrophic type usually responds very favourably to prolonged screened exposures, the treatment powerfully reinforcing Nature's attempts at the production of a curative fibrosis, causing a still greater atrophy and encapsulation of the growth, and preventing permeation of the lymphatics.

Between these two extremes—encephaloid and atrophic cancer—all modifications occur, and the prognosis depends to a very great extent upon the history and clinical features, but treatment should be conducted with the object of increasing the development of fibrous tissue, and so constricting the carcinomatous alveoli, causing degeneration of their contents, and arresting their dissemination by the lymphatics.

Very small isolated cutaneous nodules on the chest-wall, if few in number, may be treated effectively by unscreened exposures of 1-1½ hour's duration with a half-strength radium plate. If they are very numerous and closely set, prolonged exposures of 30-40 hours' duration to heavily screened rays are preferable, and this method should also be resorted to for the primary growth and infected glands, as well as for ulcerated surfaces if such exist. Superficial ulcerations unaccompanied by much

subjacent induration are peculiarly susceptible to the action of radium, and complete healing of these lesions is frequently observed. Isolated recurrent nodules, inoperable by reason of their attachment to the sternum, clavicle, ribs, or rib cartilages, are best treated by burying in them for a period of 24 hours a small powerful emanation tube screened with 1 mm. of silver.

In rapidly-growing carcinoma of the medullary type, radium can do very little except relieve pain, even though enormous doses be employed.

**Paget's disease.**—The superficial lesion in this condition is usually speedily cured by an hour's exposure to a half-strength plate unscreened, but the underlying induration and affection of glands, if present, require prolonged treatment with heavily screened applicators if any real benefit is to be obtained. In cases in which the patient is willing to submit to operation this procedure should always be adopted.

**Carcinoma of the thyroid gland.**—Considerable benefit often follows upon the employment of radium in this condition, the progress of the disease being arrested, and a decrease in the size of the growth being obtained, the general discomfort and any dysphagia which may be present being greatly relieved. Numerous powerful flat applicators should be used, screened with 2 mm. of lead, and so disposed over the surface of the growth as to obtain the maximal cross-fire irradiation. An exposure of 30-60 hours in all should be given.

It is unwise to attempt to treat the disease by the burying of radium tubes in the tumour, owing to the great tendency of these growths to fungate if any breach be made in the investing skin.

**Carcinoma of the rectum.**—The adoption of radium-therapy in inoperable cases of this disease is usually justified by the results obtained, though the amount of benefit is rarely so marked or so great as in carcinoma of the uterus. Speaking generally, the soft annular and vascular type of growth is much more favourably affected than the flat, hard, non-annular plaque with much subjacent induration. In the treatment of this latter class of growth the action of the gamma rays (which it is impossible to prevent) on the healthy rectal mucous membrane opposite the plaque produces a proctitis which, though generally transient, sometimes proves both persistent and severe, and adds greatly to the

## RADIUM-THERAPY

patient's discomfort. In the treatment of the former type, if the radium tube be of proper dimensions and introduced accurately within the lumen of the growth, little or no healthy mucous membrane is affected by the gamma rays, and no proctitis occurs. Growths situate in the upper half of the rectum appear to be more amenable to treatment than those in the lower, though this may be due to the fact that tenesmus is always more constant and severe when the growth is near the sphincters.

The performance of colostomy before radium treatment is often advisable, as the constant passage of faeces over a surface reacting to radium causes much pain. Treatment is best carried out with a 100-mg. tube screened with 2 mm. of lead and 3 mm. of rubber attached to a pliable silver wire by means of which it can be passed into the rectum and maintained in accurate apposition with the growth. A flat plate containing 80-100 mg. screened in similar fashion may also be applied over the sacrum at the level of the growth. An exposure of 30 hours' duration in all should be given, and repeated after six weeks, or longer if the proctitis has been severe. In some instances growths so treated, which were previously considered inoperable, have been so much diminished that their removal has been accomplished successfully. In other cases, where the disease is of the annular type, the carcinomatous material shrinks and is replaced by fibrous tissue, forming a stricture which may need frequent and regular dilatation.

**Carcinoma of the bladder** is often much benefited by radium. The hæmaturia, cystitis, and subjective symptoms disappear, the size of the growth is greatly lessened, and the ulcerated surfaces frequently become covered with healthy epithelium. The treatment is best carried out by the introduction of a 50-mg. tube of radium in a "window" screen of 1 mm. of silver and 1 mm. of lead inserted per urethram, and maintained by means of a pliable silver wire in such a position that the window side of the apparatus is in actual contact with the growth. If the lesion be on the base of the bladder, a second tube screened with 2 mm. of lead should be placed in the vagina in female, or in the rectum in male patients; but if on the anterior bladder wall, a flat applicator, similarly screened, should be fixed over the pubic region. The usual length of exposure is 10 hours, intravesically, in five daily applications of 2 hours

each, and 30 hours with the vaginal, rectal, or pubic applicators, spaced over the same days.

**Carcinoma of the prostate.**—The progress of this disease can generally be much retarded, and the distressing symptoms considerably relieved, by radium-therapy. When catheterization can be well tolerated, a 50-mg. tube of radium in a screen of 1 mm. of silver should be introduced per urethram, and maintained in actual contact with the growth for a period of 2 hours a day on five successive days, and its action supplemented by another tube of 100 mg. in a screen of 2 mm. of lead, introduced into the rectum for 6 hours a day on five successive days.

When catheterization is not practicable or advisable, the action of the rectal tube must be reinforced by a powerful flat applicator screened with 2 mm. of lead, and placed either on the perineum or over the pubes.

When the disease is localized to one lobe, and the enlargement of the gland is not very great, the actual burying of a silver screened radium tube within the carcinomatous mass should be adopted.

**Rodent ulcer** is, of all forms of malignant disease, the one which is most amenable to radium-therapy. For the purpose of prognosis, rodent ulcers may be divided into two definite clinical types: (1) The hypertrophic nodular type, with slight superficial ulceration of a scaly character. This class responds extremely well to radium, and yields most satisfactory results. (2) The excavating type, with undermined and overhanging edges and a gelatinous base. This not infrequently proves very intractable, and repair is most difficult to effect.

Rodent ulcers which have not previously received any active treatment and do not exceed 4 cm. in diameter are best treated by one exposure of 1½ 3 hours' duration (according to the amount of induration present) to a full-strength applicator unscreened. Although the reaction following upon this procedure is in some cases severe, and accompanied by a transient oedema of the surrounding tissues, the scars are invariably smooth, supple, and inconspicuous, and the tendency to recurrence is exceedingly slight.

When the size or position of the ulcer precludes the adoption of this method of treatment, prolonged exposures of 6, 12, 18 or more hours, with half-strength applicators screened with 0.1 mm. of lead, often prove



## RADIUM-THERAPY

most effective, and repair is obtained without any very marked local reaction.

When a mucous membrane is affected, rodent ulcer proves much more refractory, though exception should perhaps be made in regard to the palpebral mucosa, as small rodent ulcers in this situation often respond well to exposures of strong unscreened apparatus of 15-30 minutes' duration given consecutively for three days.

If the rodent ulcer has attacked bone or cartilage, great care must be exercised not to give too heavy an exposure, or a very acute, painful, and prolonged inflammation may be produced.

Many of these ulcers which have received treatment extending over a period of many years with X-rays, zinc ionization, carbon dioxide snow, etc., respond badly to radium treatment, and it is unwise to attempt to make any pronouncement as to the probable result. Quite frequently the previously treated tissues break down to an extent which far exceeds the existing ulceration, and repair is very slow and imperfect. It is often better in dealing with this class of case to depart from the routine procedure of inducing a destructive reaction by the use of a "full-strength" applicator, and to resort instead to prolonged exposures of 24 or 30 hours' duration, with half-strength applicators, screened with 2 mm. of lead. This method of treatment should invariably be adopted in those distressing cases (now happily becoming very rare) which have existed for ten, fifteen or twenty years, have destroyed the greater portion of the face, and invaded its cartilaginous and bony tissues. "Cure" in these cases is impossible, but a very great deal can be done to arrest the progress of the disease, lessen the foul discharge, and render the patient much more comfortable.

**Sarcoma.**—Speaking in general terms, sarcomata, if taken in their early stages and before dissemination has occurred, do very well under radium treatment. The burying within the growth of tubes of radium salts or radium emanation should be resorted to whenever practicable, and the action of these tubes may be much helped by the external application of radium plates heavily screened. The screening of the tubes should not exceed 1 mm. of silver, as the employment of thick lead or platinum screens is often followed by necrotic changes in the cells in their immediate neighbourhood, and a sinus forms and remains open for a very long time.

It is essential to give as vigorous a treatment as possible, as the great vascularity of these growths, and their rapid and wide dissemination by the blood-stream, are factors which militate very strongly against the chances of success.

The best results are obtained in sarcomata of the tonsil and postnasal space, and the effect in many such cases is very striking, the growths completely disappearing within six weeks of the treatment.

Lymphosarcomata also give excellent results.

**Lymphadenoma.**—The most striking feature in the treatment of this disease with radium is the extreme rapidity with which the size of the affected glands is diminished, the effect being often clearly perceptible to the patients themselves within a week of the first exposure. To obtain this result the employment of numerous large and powerful applicators, heavily screened, is necessary, and this heavy dosage is almost always followed by severe systemic disturbance, with high temperature and general malaise. After the reaction has subsided, considerable improvement is generally apparent, though in the majority of cases recrudescence occurs.

The best results are obtained when the condition is confined to the lymphatic glands—the spleen not being affected.

**Mediastinal tumours.**—In the majority of instances the rate of growth is very greatly checked by the use of radium, and occasionally a definite decrease in the size of the tumour, as seen by X-ray screening, ensues.

If the condition be accompanied by an accumulation of fluid in the pleura, and paracentesis be performed, radium appears to exert a very remarkable action in lessening the tendency to the production of further effusion. In these cases the use of a very large quantity of radium (400 to 500 mg.) is indicated. The applicators should be screened with 2 mm. of lead, and disposed in such fashion over the chest-wall as to procure the maximum "cross-fire" irradiation.

Treatment must be very persistent, a total exposure of not less than 30 hours should be given, and the series repeated at the end of four to six weeks. This heavy dosage is almost invariably followed in two or three days by a feeling of great fatigue and exhaustion, which, however, passes off completely in about a week's time.

**Fibroid disease of the uterus.**—Radium exerts a most beneficial action upon the distressing

## RADIUM-THERAPY

symptoms of menorrhagia and metrorrhagia, which are the almost invariable concomitants of this condition, and occasionally produces some diminution in the size of the uterus. As the cervical canal is rarely sufficiently patulous to admit a screened radium tube without previous dilatation, and patients are often averse from this, the treatment may be carried out by the introduction of a tube containing 100 mg. of radium, screened with 2 mm. of lead, into the posterior fornix, and the application of a plate of from 80–100 mg. similarly screened over the fundus uteri. The total exposure should be 30–60 hours, spread over from five to ten days, and the series should be repeated after three or four months. The first effect noted is the checking of the metrorrhagia, and this is followed by a progressive decrease of the menorrhagia until the flow becomes normal, or sometimes ceases altogether. If any leucorrhœa exists, this, too, is much diminished in amount, and not infrequently completely disappears.

**Nævi (flat superficial nœvi, capillary nœvi, "port-wine stains").**—If blanching be readily effected by gentle diascopic pressure, the result of the treatment will probably be satisfactory; but if it be found impossible to produce blanching except by great pressure, it is unlikely that radium can do much for the condition.

The factor of personal idiosyncrasy is always a prominent one, and it is exceedingly difficult to lay down any rules as to the strength and duration of exposures.

Infinite patience is necessary in the treatment of superficial nœvi, and the utmost caution must be observed. The effect of each exposure should be studied most carefully, as too frequent treatments or excessive dosage may result in the production of troublesome telangiectases. It is best to use quarter- or half-strength applicators screened with 0.01 mm. of aluminium, and to start with short exposures of 15–20 minutes' duration, gradually increased until a satisfactory reaction has been obtained. So soon as the lesion has assumed a salmon-pink colour, the screening should be altered to 0.1 or 0.2 mm. of lead, and the length of exposure increased to 1, 1½, or 2 hours.

The best results can be looked for when the nœvus is quite superficial, and shows no tendency to infiltration. If such infiltration exists the treatment will have to be much more vigorous, and a destructive reaction of slight

degree produced. A smooth, supple, and white scar will be left.

**Cavernous nœvi.**—These do excellently under radium, especially when of such shape that "cross-fire" radiation is possible. Half-strength applicators screened with 0.1 mm. of lead should be employed, and the exposure should be of 1–3 hours' duration, spread over three successive days. Little or no surface reaction is produced by this method of treatment, and the nœvus shrinks gradually and steadily. If, on palpation, the presence of pulsating vessel is appreciable, the case is not likely to prove completely successful, unless the vessel first be ligated.

**Warts and other papillomata** yield readily to exposures, with half-strength apparatus unscreened, of 30 minutes' to 2 hours' duration, according to the size and thickness of the lesion. The reaction is but slight, and the resultant scar scarcely noticeable.

**Tuberculosis of glands.**—In cases in which surgical measures have been declined for cosmetic reasons, radium often proves of considerable value, either when used alone or as an adjunct to vaccine treatment. Heavily screened exposures of 30 or more hours' duration should be resorted to and an effort made to secure "cross-fire" irradiation where practicable. If the glands be caseating, radium treatment is of very little use.

**Lupus vulgaris.**—As a routine treatment for this disease Finsen light is greatly to be preferred to radium, but where it fails to effect a cure or improvement, radium sometimes proves of great use. Unscreened exposures of 1–2 hours' duration must be given and a destructive reaction produced. The resultant scar is usually firm, smooth, supple, and inconspicuous.

**Spring catarrh.**—In this condition the employment of radium is strongly to be advocated, as it will sometimes cure the most intractable cases. Personal idiosyncrasy is, however, a very important consideration, and it is essential to proceed with great caution, giving short unscreened exposures at intervals of a fortnight, and carefully noting the reaction. If the dosage be adjusted accurately, the granulations on the palpebral conjunctiva gradually disappear, there is no severe inflammation, and no resultant scarring of the mucous membrane. A special form of applicator should be used which can be introduced readily into the conjunctival fornices. It should be of full strength, and the first

## RADIUM-THERAPY

exposure should not exceed 15 minutes' duration. No screening is employed. If the effect be not satisfactory, the length of the exposure may be increased to 20, 25, or 30 minutes until the desired result is obtained.

**Cheloid.**—This affection of the skin usually gives most excellent results when treated with radium; and a great improvement, if not complete cure, can safely be predicted when the condition is of recent origin and occurs in young subjects. In tender and painful cheloids the anæsthetic effect of radium is generally very pronounced and appears early in the treatment.

Half-strength applicators, screened with 1 mm. of silver, should be employed, and an exposure of 18–30 hours spread over from three to five days, according to the size of the lesion, given. This brings about a gradual absorption of the cheloid unaccompanied by any surface irritation. If, however, time be of importance, or the cheloid prove refractory, screening of 0.5 mm. silver or 0.1 of lead should be employed, though the reaction will probably be very definite and accompanied by vesication.

**Lupus erythematosus.**—In early cases of this disease which have not been subjected to much other treatment, one or two exposures not exceeding an hour in duration with a half-strength apparatus unscreened or screened with 0.01 mm. of aluminium may suffice to bring about the disappearance of the lesion. Care should be taken to ensure that the applicator employed is sufficiently large to reach well beyond the borders of the patch treated.

**Psoriasis and chronic eczema.**—Both these conditions generally yield readily to short unscreened exposures of 2–5 minutes' duration, given on three successive days, the series of exposures being repeated at intervals of a fortnight. The patches quickly disappear after a slight superficial reaction, and in eczematous cases there is but little tendency to recurrence. With psoriasis, however, the disease almost invariably reappears after a time, though with some patients the interval that elapses may be of many months' duration.

**Lichenification of the skin,** with its intolerable itching, is quickly relieved and often completely cured by one exposure of 10 or 15 minutes' duration to a half-strength applicator, screened with 0.01 mm. of aluminium.

**Pruritus.**—The analgesic effect of radium is often of the greatest use in this affection, and short unscreened exposures frequently produce a degree of relief which is unattainable by any

other measures. This is especially noticeable if the pruritus occur in association with a definite lesion such as leucoplakia or hyperkeratosis. If the dosage be carefully considered and treatment repeated at fortnightly intervals until the skin is restored to its normal condition, complete and permanent cure may result. If, however, the condition be a neurosis, the prognosis is not so hopeful, and the benefit derived, if any, is not likely to be of very long duration.

**Exophthalmic goitre.**—The results obtained by radium-therapy in this condition are sufficient to justify its adoption, when routine treatment has failed to bring about any improvement. Prolonged exposures of 30 or more hours' duration in all should be given, with heavily screened apparatus containing 200 or 300 mg. of radium, the apparatus being so disposed as to cover as much as possible of the gland surface. The patient should be warned that the first effect of the treatment may quite possibly be an exacerbation of all the symptoms, lasting perhaps for two or three weeks. This phenomenon is undoubtedly occasioned by the radium reaction producing an increased influx of the thyroid secretion into the blood-stream; the disturbance, however, gradually passes off, and definite improvement slowly manifests itself. In very susceptible patients one series of exposures may be all that is required; in others, treatment may have to be repeated at intervals of three or four months. In very advanced cases, associated with extreme tachycardia, great wasting, frequent vomiting or diarrhoea, results are not nearly so good. The treatment has to be undertaken with great caution, and the effects carefully watched, as if the dosage be at all excessive, harm may result.

**Rheumatoid arthritis and osteo-arthritis.**—The daily administration of 250 c.c. of radium emanation solution, of a strength of not less than 1 millicurie per litre, in these painful and crippling diseases is sometimes attended by remarkable results. The cases which appear to derive most benefit are those in which the disease is of relatively short duration, and the changes are periarticular in type and multi-articular in distribution. The patient's age also exerts some influence, those under 40 responding more quickly to the action of the emanation. Cases with a definite gouty history sometimes experience an acute exacerbation of all their symptoms within the first week or ten days of treatment, but this dis-

## RAPE

turbance is quite transient, and is usually followed by a distinct change for the better.

Little or no improvement can be looked for in instances where cartilaginous or osseous changes are predominant, and the radium emanation solution is powerless to bring about the absorption of osteophytic growths or the loosening of bony ankylosis. When, however, limitation of movement is due to periarticular fibrous thickening, considerable increase of mobility often follows on the taking of the radium water. Other favourable effects are that the muscular and articular pains are lessened or disappear, the grating of the joints on movement is not so observable, the muscles regain much of their lost tone, and the patient's general health is greatly improved. This is especially noticeable in cases where anæmia is present, the hæmoglobin index being rapidly increased. The treatment must, however, be persisted in for quite a long time, and at least six weeks are likely to elapse before any change is noted.

**Prophylactic postoperative irradiation** with the ultra-penetrating rays from heavily screened apparatus is often of considerable value in preventing or minimizing the danger of recurrence after operations for the removal of carcinomata or sarcomata, and is of especial service in those cases of malignant growth in which it has been found impossible to operate well beyond the appreciable area of the disease.

A. E. HAYWARD PINCH.

**"RAILWAY SPINE"** (*see* NEUROSES, TRAUMATIC)

**RAPE.**—Rape is defined in law to be the carnal knowledge of a woman by force and against her will, and any person convicted of the crime is guilty of a felony. The crime can be committed on the chaste as well as the chaste. The Criminal Law Amendment Act, 1885, lays down certain conditions regarding age, mental condition, etc. By this Act any person who has carnal knowledge of a girl under 13 years (with or without her consent) is guilty of felony, while the attempt is a misdemeanour. Any person who has or attempts to have carnal knowledge (even with consent) of a girl above 13 but under 16 years of age is guilty of a misdemeanour; but if it be made to appear to the court or jury that the accused had *reasonable* cause to believe that the girl was of or above 16 years, then consent must be accepted as a sufficient defence. Any person who has or attempts to have carnal

knowledge of a female idiot, or lunatic, or imbecile, in circumstances which do not amount to rape, but which prove that the offender knew that the girl or woman was an idiot, or lunatic, or imbecile, is guilty of a misdemeanour.

If consent to connexion be obtained by personating a husband, the offence is rape.

The administration of any drug, matter, or thing to a female with intent to stupefy or overpower, so as to enable any person to have unlawful carnal connexion, is a misdemeanour.

Penetration of the penis within the vulva is all that is necessary to constitute rape. The hymen need not be destroyed, nor is emission necessary.

From the foregoing statements of law it will be obvious that medical evidence will relate chiefly to connexion and violence, the question of rape being one of fact for the jury.

**Examination.**—Where possible, both the victim and the accused should be examined, but it must be remembered that no authority can compel a person to submit to examination against his or her will. Consent to make the examination should be asked for in the presence of one or more disinterested witnesses, and it should be plainly stated that any information obtained may be used against as well as for the person examined. In the case of children the consent of the parent or guardian should be obtained.

Note should be made of the date and hour of the examination and of the alleged rape, together with the names of the witness or witnesses present when consent was given and at the examination.

In the **female**, attention should be directed to the general physical appearances; tears and stains (blood, seminal, mud, etc.) of clothing; marks of violence on parts of the body other than pudenda; the condition of the genitals with regard to signs of virginity or defloration, marks of violence, presence of blood and semen, signs of venereal disease.

The physical appearance of the woman is important, especially when compared with the accused, as it is expected that a woman in full possession of her senses will resist to the limits of her power. The presence of scratches, bruises, and other marks of violence, together with tearing or disarrangement of the dress, is evidence of a struggle. The possibility of a healthy adult female being overcome by one man, without evidence of violence being found, is remote, and special care should be taken in

## RAPE

investigating these cases. Apart from the possibility of the woman having been drugged or having fainted, she may have been rendered incapable of resistance by terror. Disproportion in age and strength between victim and assailant is similarly of importance; a feeble old woman cannot be expected to resist a strong man.

Generally speaking, marks of injury will be found on the genitals of children where the crime has been completed, whereas in adult females accustomed to copulation the *act of connexion itself* will leave no injuries on the genitals, though scratches by the finger-nails may be found on the external genitals, and marks of violence on other parts of the body. The crime of rape may be committed on an adult virgin without injury to the hymen.

The signs usually relied upon to establish virginity are these:—

**Breasts.**—The nipples are usually small and undeveloped, and the areola is pink or slightly darker according as the woman is blonde or brunette. As the condition of the breasts changes only with pregnancy, their appearance is no indication of a single coitus.

**Hymen.**—This may have ruptured as the result of ulceration or accident, and in certain rare cases is congenitally absent. It is present in the great majority of virgins, and in form varies from a slight annular ring to a membrane completely closing the entrance to the vagina. Usually one or more openings, varying in size, shape, and position, give entrance to the vaginal canal. The membrane itself may be thick and resistant, or thin and elastic. While absence of the hymen does not always indicate loss of virginity, neither does its presence always point to non-intercourse, as women have been known to conceive with the hymen uninjured.

**Vagina.**—In women who have not had intercourse the canal is narrow and the walls are rugose, but this condition is not likely to be affected by a single connexion.

**Fourchette and perineum.**—The fourchette may be ruptured by the first connexion, and rupture of the perineum has been reported but is not common.

Blood is not always present after defloration, but may be excessive where the hymen is thick and vascular. When blood is found about the genitals, the hymen (and other parts of the vulva) should be examined for laceration, as the blood may be due to menstruation and not to injury. The vulva, pubic hairs, and

clothing should be examined for semen (*see SEMINAL STAINS*). Fluid is best removed from the vagina by a glass pipette having a rubber teat attached, and microscopic slides should at once be spread with the fluid removed and fixed by gently heating over a Bunsen or spirit flame.

**Venereal disease** not infrequently follows rape on young girls; in fact, the reason for the crime is often the vulgar belief that connexion with a virgin will cure venereal disease. Purulent discharge from the vagina is not necessarily a sign of venereal disease, but may be due to simple vaginitis arising from local irritation, as from uncleanness or worms. Where syphilis or gonorrhœa is discovered, regard must always be had to the factor of time. In gonorrhœa the period of incubation is usually two to four days, but may be as long as a week. In syphilis the primary sore appears in from two to six weeks (generally about three) after infection. Again, either disease may be acquired in other ways than by sexual connexion. In young children gonorrhœa has frequently been communicated by unclean sponges, towels, etc. In syphilis the site of the primary sore is of importance, e.g. on genitals after sexual connexion, on lips after kissing, etc.

In the diagnosis of venereal disease, laboratory as well as clinical methods should be employed and the bacteriological examination carried out by one skilled in laboratory technique. Medium containing blood-serum (e.g. Martin's or Thomson's) should be employed, and the tubes should be inoculated direct from the discharge without loss of time. In sores which are clinically syphilitic, confirmation may be obtained, in the case of the primary sore by microscopical examination for the *Spironema pallidum*, using the fresh serous exudate and dark-ground illumination, and by the Wassermann reaction in both primary and later lesions.

**Examination of the accused.**—The examiner should look for scratches on face, hands, legs, etc.; stains (blood, seminal, fluid, etc.) on clothing and person; condition of genitals (wounds and venereal disease). Rupture of the frænum penis may be found, and indicates the forcible introduction of the penis into an opening comparatively small. Men showing no clinical evidence of gonorrhœa may, as the result of past disease, still have active gonococci and be capable of conveying infection.

**Violation before death.**—When the dead

## RAT-BITE FEVER

body of a female has been discovered the medical examiner may be required to state if the deceased had been violated before death. Special attention should be paid to marks of violence on the genitals and body generally, as already described. Even the presence of spermatozoa in the vagina will merely prove that coitus has occurred, and not that rape has been committed.

A. ALLISON.

**RAT-BITE FEVER.**—Recognized for centuries in China and Japan, it is only comparatively recently that reports show that rat-bite fever, though a rare disease, is widely distributed. Amongst others, Horder and Box have described cases in England.

The initial bite may heal slowly, but no constitutional disturbance may be noted for some weeks, or even months. After this incubation period, averaging four or five weeks, the patient is taken suddenly ill with shiverings and perhaps rigor, and his temperature rises rapidly to 104° F. or even higher. There may be pains in the muscles and joints, and delirium. Before or synchronously with the fever, local reaction may occur in the wound, which, after becoming red, swollen, and tender, may ulcerate, while the lymph-glands which drain it also show inflammatory changes. The febrile attack lasts three or four days, and ends as suddenly as it began, the patient appearing quite well again. Recurrences follow, however, at varying intervals, in some cases daily, in others as infrequently as every two or three weeks, or even longer. Each febrile attack is accompanied by a rash, either erythematous or purplish and resembling that of measles. There is a moderate leucocytosis. The succession of attacks may persist for months and, rarely, for years. According to Hata, the mortality is 10 per cent.

The infection is probably a spirochaetosis, as has been shown by Futaki; a sporozoan parasite, a streptothrix, and a bacillus have each been incriminated.

**Treatment.**—The wound should be cauterized in all cases of rat-bite as a preventive measure, and this procedure is also the first step in treatment. An arsenobenzol compound, if given early, may abort the disease.

FREDERICK LANGMEAD.

**RAYNAUD'S DISEASE.**—Raynaud's disease, or symmetrical gangrene affecting the extremities, is common in women in early adult life. Numerous causes have been sug-

## RAYNAUD'S DISEASE

gested as contributory to its development, such as exposure to cold, worry, fright, and general nervous depression. At first the attacks are paroxysmal and almost invariably limited to the cold months of the year, though ultimately even the summer months do not bring immunity. The attacks begin with paroxysmal blanching of the fingers or toes, which become waxlike and very painful, evidently due to arterial vaso-constriction. After a time the extremities become dusky and cyanosed and very cold. The attack may pass off, or the cyanosis may deepen in colour to black, and patchy gangrene may occur on the tips of the fingers or toes. There is never the severity of gangrene in this condition which may be met with in chronic obliterative endarteritis, anything more than slight loss of tissue of the fingers and toes, or of the extremities of the ears, rarely occurring.

**Diagnosis.**—The stage of local syncope of the extremities must be distinguished from a similar condition known as "*dead fingers*" to which many people are liable without development of the more severe condition of Raynaud's disease. Such attacks of local syncope are sometimes associated with paroxysmal hæmoglobinuria. Another disease which must be distinguished from Raynaud's is *chronic ergotism*, a condition in which the pain and gangrene are much more severe than in Raynaud's disease. *Erythromelalgia* and other vaso-motor neuroses are usually easily differentiated. In addition, many chronic nervous diseases may cause some cyanosis and gangrene of the extremities, such as tabes, syringomyelia, peroneal atrophy, and disseminated sclerosis, as well as diabetes and other causes of multiple neuritis.

**Treatment** should include attention to the general health and nutrition. The limbs should be kept warm during the winter months, and exposure to cold avoided as far as possible. Local treatment by galvanism and hot-water baths is often useful, but over-stimulation by faradism or massage is rather to be avoided. The disease is not a progressive one like obliterative endarteritis, and the prognosis is on the whole good, though it is apt to be very chronic for years, with liability to some loss of tissue at the periphery of the extremities as a sequela.

WILFRED HARRIS.

**REACTION OF DEGENERATION (see ELECTRICAL REACTIONS).**

## RECTAL FISTULA

**RECKLINGHAUSEN'S DISEASE** (*see* SKIN, FIBROMATA OF).

**RECTAL FEEDING** (*see* GASTRITIS, ACUTE; ENEMATA).

**RECTAL FISTULA.**—This condition is mentioned under the head of ANAL FISTULA (q.v.). The majority of fistulae which follow ischio-rectal abscess are limited to the anal region, but occasionally they track up under the mucous membrane of the rectum; sometimes they result from abscesses forming above a rectal stricture, in which case the sinus will track up to a point above the stricture. A rectal fistula as a result of gunshot wound of the sacral region may give rise to trouble. In such cases the bowels must be kept freely open and every attempt made to procure healing of the sacral fistula. Failing natural healing, a plastic operation will be necessary.

ZACHARY COPE.

**RECTOCELE** (*see* PELVIC ORGANS, FEMALE, DISPLACEMENTS OF).

**RECTUM, INFLAMMATION OF** (*see* PROCTITIS).

**RECTUM, INJURIES TO.** The rectum may be injured in various ways: (1) There may be direct injury by a sharp or a blunt object, such as would result from falling on to a spiked railing or sitting down on a sharp object. The object may enter the anal canal and travel several inches up so that serious damage may ensue. (2) The injury may be indirect, consequent on fracture of the sacrum and coccyx or of the lateral portions of the pelvis resulting from crushes, etc. (3) Gunshot wounds may traverse the rectum from any direction. (1) Some cases have been recorded in which the nozzle of a compressed-air apparatus has been introduced into the anal canal with consequent rupture of the colon or rectum owing to gaseous distension.

**Signs and symptoms.** *Shock* is a constant accompaniment of rectal injuries caused by severe trauma, but varies considerably in degree. *Bleeding* from the rectum will result from tears of the gut-wall. If the rectum be torn at the level of or above the peritoneal reflection, *peritonitis* may ensue. Then abdominal pain, vomiting, and hypogastric rigidity soon develop. Other complications of the original injury may accompany tears of the rectum, e.g. *ruptured urethra* or *bladder*, or *fractured sacrum*. Severe rectal tears are some-

## RECTUM, MALFORMATIONS OF

times accompanied by *prolapse of the small intestine* into the perineum.

**Diagnosis.**—There is usually no difficulty in diagnosing an injury to the rectum if care be taken in examination. In fractures of the pelvis the rectum must carefully be examined digitally. When a deep perineal tear is present any rectal injury will be detected while the patient is under the anaesthetic given for the purpose of suturing the wound. If there is any prolapse of small intestine the seriousness of the peritoneal injury will be evident, and the onset of peritonitis will also point to the likelihood of a similar lesion.

**Treatment** in slight cases is directed to keeping the injured part as clean as possible so as to prevent infection of the perirectal tissues. Any external anal tear must be sutured. In more serious cases with evidence of peritonitis, or complications such as ruptured urethra, surgical intervention will be necessary.

ZACHARY COPE.

**RECTUM, MALFORMATIONS OF.**—The rectum and anal canal are developed from different structures. The anal canal is formed by the hollowing out of an epithelial plug formed by proliferation of the posterior part of the cloacal membrane. The rectum forms the posterior end of the hindgut, which during the second month opens into the cloaca, but becomes separated from the uro-genital sinus by the uro-rectal septum and the perineal septa.

The common malformations of the rectum and anus are imperforate anus and maldevelopment of the rectum.

(1) **IMPERFORATE ANUS.**—In this case the cloacal membrane fails to break down so as to bring the rectum into communication with the exterior. Sometimes there is only a slight depression in the anal region, whilst at other times the anus is well formed and is only separated from the rectum by a thin membrane at the bottom of an anal pit.

(2) **MALDEVELOPMENT OF THE RECTUM.**—The rectum may not extend backwards so far as it normally should, and there may be a deficient development of the perineal septa, so that the uro-genital part of the cloaca is not completely separated from the rectal part. In such cases the rectum may communicate with the prostatic urethra in the male or may open at the posterior margin of the vagina in the female. These conditions are usually accompanied by imperforate anus.

## RECTUM, NEW GROWTHS OF

**Diagnosis.**—It is usually easy to diagnose an imperforate anus. It is often noticed at birth that there is no depression in the anal region. When meconium does not come away during the first twenty-four hours a finger inserted into the anus will detect the obstruction due to the imperforate cloacal membrane.

When the rectum communicates with the prostatic urethra, faeces come away with the urine. In the female, when the rectum opens at the posterior margin of the fourchette the urine and faeces may appear to come from a common cloacal orifice, since the anterior part of the genito-urinary region is also ill developed.

**Treatment.** When the anus is imperforate and the hindgut has no other outlet, operation must be undertaken as soon as possible. If there is an anal depression and it can be made out that there is but a thin membrane separating the exterior from the rectum, the septum should be freely incised. Bulging on inspection or resonance on percussion are indications that there is but a thin partition.

When there is no indication as to how distant the rectum is from the anus, special surgical advice should be sought, since it may require a deep incision to find the rectum, and in exceptional cases a colostomy may be required, or at least an abdominal section may be needed to find the lower end of the hindgut.

When there is an approximation to a cloacal condition and the rectum and vagina and possibly the urethra appear to open into a common space, there is no urgency for any operative procedure, since there is no likelihood of intestinal obstruction.

When faeces come away via the urethra, a plastic operation may be necessary in addition to treating any accompanying imperforate anus.

ZACHARY COPE.

**RECTUM, NEW GROWTHS OF.**—The only common **simple** rectal growth is an **adenoma**, though cases of angioma, papilloma, fibroma, and even lipoma have been recorded. The adenoma may be single, as it commonly is in children, or multiple. In the latter case there are usually similar adenomata in the colon, and the condition is more serious from the tendency to recurrence after removal.

The only **symptom** of importance is hæmorrhage, which is usually an accompaniment of adenoma, and may be caused by an ulcerated

papilloma or angioma. When an adenoma has a long pedicle it may prolapse on defæcation.

**Diagnosis** can be made by use of the proctoscope and by digital examination. The absence of any tendency to infiltrate the subjacent tissues will serve to distinguish the growth from malignant disease.

**Treatment.**—Adenomata and fibroid polypi and papillomata should be removed. This is usually a simple operation, but before removing multiple polypi the condition of the colon must be investigated.

Of **malignant** rectal growths, sarcoma is uncommon, carcinoma common. The symptoms are somewhat alike. Rectal **cancer** is usually a columnar-celled growth. It takes the form of an irregular mass projecting into the rectum, of an ulcerating surface with infiltrated edges, or of an infiltration of the rectal wall causing a complete circular stricture. The growth spreads outwards into the ischio-rectal fossæ and backwards towards the sacrum: the glands in front of the sacrum become affected. Later, secondary deposits may be found in the liver.

**Symptoms.** These may be grouped as due to (1) irritation and ulceration; (2) obstruction; (3) extension to neighbouring structures; (1) the effect on the general health.

(1) A discharge of blood and mucus is often the only symptom of which complaint is made. The blood is at first only slight in amount and chiefly noticed on defæcation. The excess of mucus is due to the irritation of the glands of Lieberkühn. Later in the course of the case bleeding may be more severe, and the frequent passage of bloodstained mucus may simulate diarrhoea.

(2) Sometimes acute intestinal obstruction is the first serious symptom, but inquiry usually elicits that constipation has been a complaint for some weeks or months previously. Constipation of itself is not of serious significance, but in one whose bowels have previously been regular it should always lead to a rectal examination. Constipation is never severe in an early stage of the cancer, though it may be one of the earliest symptoms, but to cause any appreciable narrowing of the rectum a considerable mass of growth is required.

(3) As the growth extends through the bowel-wall it obstructs the hæmorrhoidal veins and causes piles, which are occasionally an indication of cancer of the large bowel. When extension has proceeded into the ischio-rectal fossæ or the presacral space, pain is complained



## RECTUM, PROLAPSE OF

of. Pain is a *late* symptom, and usually indicates an inoperable or barely operable growth. In the male, extension anteriorly towards the prostate and base of the bladder may cause frequency of or pain during micturition.

(4) By the time serious constitutional symptoms have appeared, rectal cancer is usually far advanced, but there is often a slight general depreciation of health at a much earlier stage. It must be remembered that during the operable stage of many cancers of the rectum the patient appears to be in good health. When anæmia and loss of weight make their appearance the outlook is not promising.

**Diagnosis.**—In view of the frequency of cancer of the rectum and the lateness at which it is so often diagnosed, it should be an invariable rule that in cases with rectal symptoms or increasing constipation a digital rectal or sigmoidoscopic examination should be undertaken. *This is especially necessary in the case of piles, for many cases of cancer are treated as piles until the more serious symptoms show that there is a more serious disease.* The cancer is usually readily diagnosed by palpation. The irregular friable surface projecting into the bowel is characteristic. *Fibrous stricture* is distinguished by the firmness and lack of friability of the tissues and the slower progress of the symptoms, apart from the appearance on sigmoidoscopic examination. *Diverticulitis of the sigmoid* may cause symptoms very similar to those of rectal cancer; the discharge of blood and mucus and severe wasting may be evident. It may need an abdominal exploration to distinguish one from the other.

The **treatment** is usually operative, either by excision or by colostomy. The application of radium sometimes benefits temporarily (see RADIUM-THERAPY).

ZACHARY COPE.

**RECTUM, PROLAPSE OF.**—There are two main varieties of rectal prolapse—(1) that in which only the mucous membrane comes outside the anus, (2) that in which the whole thickness of the rectal wall prolapses.

**Etiology.**—Anything which causes weakening of the supports of the rectum or which induces straining may predispose to prolapse, e.g. constipation, severe diarrhoea with tenesmus, stone in the bladder and urethral obstructions, loss of weight and debility consequent on advancing years, etc.

**Symptoms.**—Prolapse of the rectal mucosa leads to a constant wetness of the perianal region due to the secretion of mucus from

the irritated protruded portion. The external sphincter is weak. The patient complains of local discomfort and something "coming down." Examination shows a projection of mucous membrane extending 1-2 in. outside the anus. The projected portion has the normal colour of mucous membrane, is not lobulated, nor does it show venous masses as do prolapsed internal piles.

Prolapse of the whole thickness of the rectum occurs chiefly in young children or in old, debilitated persons. The protruded portion may be of considerable size, tends to have the opening towards the back of the apex, and if exposed for long may have ulcerated patches on the exposed mucosa.

**Diagnosis** between simple prolapse of the mucous membrane, which never projects more than an inch or two, and prolapse of the whole bowel has to be made. From a protruded *intussusception* the diagnosis is made by noting that there is no groove between the muco-cutaneous junction and the prolapsed mass. In *intussusception* there are acute abdominal symptoms, and the finger can be inserted into the anal canal between the protruded mass and the external sphincter.

**Treatment.**—In slight cases of prolapse of the mucous membrane the condition may be improved by preventing constipation. An ounce of olive oil may be injected into the rectum overnight, and liquid paraffin may be administered by the mouth. Whenever the parts come down they should be replaced. In persistent cases ligature of the prolapsed portion is necessary.

In prolapse of the whole thickness of the rectum it is necessary to treat any exciting cause such as diarrhoea or constipation, and to interdict the adoption of a bad posture during defæcation. In the case of a young child the motions may be passed on to a mackintosh with the child lying on its side. Generally speaking, it is better for the patient to defæcate squatting on a low pan rather than sitting on a high seat. The bowel should always be replaced when it comes down. When the portion protruded is great, replacement must be done by gentle pressure with a warm sponge or pad of cotton-wool, starting at the apex of the mass.

In most cases of severe prolapse in adults some form of operation will be necessary, but in children the condition is often temporary and improves with the general health.

ZACHARY COPE.

## RECTUM, STRICTURE OF

**RECTUM, STRICTURE OF.**—Rectal stricture may be (1) congenital, (2) due to scarring or inflammatory conditions, (3) malignant. (1) *Congenital* stricture occurs in the region of the muco-cutaneous junction, and is due to imperfect disappearance of the anal plug. The narrowing is easily felt by the examining finger. (2) Stricture may occur after operation upon the rectum, especially for hæmorrhoids. It is wise to examine the rectum a fortnight after an operation for piles in order to make sure that no narrowing of the canal has occurred. Stricture develops also after ulceration of the rectum or inflammatory conditions in the rectal wall or affecting the neighbouring tissues. In women pelvic cellulitis may constrict the rectal canal. Syphilis, gonorrhœa, and dysentery have all been stated to be causes, but in the majority of cases it is impossible to discover any specific cause for the formation of a fibrous stricture. (3) *Malignant* stricture is considered elsewhere (p. 85).

**Symptoms.**—*Congenital* stricture may cause constipation, but usually there is no other symptom of importance. *Fibrous stricture* leads to gradually increasing constipation, often alternating with attacks of spurious diarrhœa. The irritation on the proximal side of the stricture frequently leads to ulceration, and to the formation of perirectal abscesses and fistulæ. A discharge of mucus mingled with a little pus or blood is sometimes noted.

**Diagnosis.**—Digital examination of the rectum will always reveal the stricture. The narrowing may be slight and annular, so that the finger may pass through it, but more commonly the canal is thickened and fibrosed for an inch or more, and the finger can then only engage in the lower end of the stricture. Examination with a long proctoscope or the sigmoidoscope will show the lower entrance to the stricture. Careful rectal examination must be made in every patient with anal fistulæ to ascertain if these are secondary to stricture. Diagnosis between fibrous and malignant stricture is usually easy. With the latter, there is usually a greater amount of bleeding, and examination by the finger reveals an irregular mass projecting into the bowel lumen. The irregularity and friability of the mass and the tendency to infiltrate the neighbouring tissues distinguish cancerous from fibrous stricture.

**Treatment.**—In slight strictures low down in the rectum the passage of rectal gum elastic bougies may be sufficient to keep the canal

## REFLEXES

sufficiently patent. With long or very narrow strictures some form of operative interference is usually indicated. This may be posterior incision followed by the passage of bougies, or excision of the whole stricture, whilst in very bad cases with many fistulæ colostomy may be necessary.

ZACHARY COPE.

**RECURRENT VOMITING** (*see* VOMITING, CYCLICAL).

**RED DEGENERATION** (*see* UTERUS, NEW GROWTHS OF).

**REFERRED HEADACHES** (*see* HEAD-ACHE).

**REFLEXES.**—The reflexes are of great importance in clinical neurology, as alterations in them are the most delicate tests we possess of disturbances of certain nervous functions, and particularly since they are objective signs which do not require the co-operation of the patient, as most of our other tests do.

There are two separate classes of reflexes, viz. the deep reflexes or tendon-jerks, and the cutaneous or superficial reflexes.

**The tendon-jerks.**—If the tendon of any muscle that is in a normal state of tone be suddenly tapped in such a way that it stretches the muscle, this muscle responds by a sharp brisk contraction after a very short interval of time. Such a reflex contraction can be obtained from every muscle that can be suddenly elongated, by a tap on its tendon or on a movable structure to which the tendon is attached. These tendon-jerks were formerly regarded as manifestations of muscle-tone—it was believed that muscles in a normal state of tone responded directly and without the intervention of a reflex arc—but it has been now shown that they are true spinal reflexes and are subserved by an arc consisting of afferent fibres that carry impressions from the muscles, a reflex centre in the grey matter of the cord, and an efferent limb that passes through the ventral roots and the motor nerves to the muscles. (*See* NERVOUS SYSTEM, PHYSIOLOGY OF.)

The tendon-jerks that are most commonly tested in clinical work are the knee-jerk, the ankle-jerk, and the flexor and extensor jerks of the arm.

The **knee-jerk** may be elicited as the patient sits on a chair with his knees semiflexed and his feet resting on the floor, or as he sits on a high stool with his legs hanging vertically; the more common position in which the limb to be tested is crossed over the opposite knee

## REFLEXES

is not so satisfactory. Then the patellar tendon is struck firmly with a suitable percussion hammer or with the ulnar border of the observer's hand. If the reflex is present a sharp twitch contraction of the quadriceps extensor extends the knee. Either the contraction of the muscle or the movement that it produces may be observed.

The knee-jerk is usually regarded as the easiest of clinical signs to demonstrate, but it is not so. For it is essential that the limb be properly relaxed, and it is often difficult to obtain this or to make sure of it. I have frequently seen patients in whom the jerks were wrongly believed to be absent because proper relaxation of the muscles was not obtained; this is particularly liable to happen after the muscles have been tested repeatedly, especially in nervous persons and in compensation cases. If there is any doubt, the left hand should be placed in the popliteal space, where it can feel the tendons of the hamstrings; in fact, one of the most satisfactory ways of demonstrating the reflexes is to place one hand behind the knee and raise it till the joint is flexed to an obtuse angle as the patient lies on his back. Or the patellar tendon may be tapped unexpectedly, or when the patient's attention is diverted, as he lies on his side with his knees semiflexed.

The knee-jerks are invariably present in health, and their absence is always evidence of some organic nervous trouble, or of a functional disturbance in their reflex arc—that is, of either the afferent or efferent fibres of the arc, or of its spinal centre, which lies in the third and fourth lumbar segments. Absence does not necessarily mean structural change; it may be due to a functional depression, as it often is immediately after the onset of an acute hemiplegia or after transverse section of the cord. Diminution of the jerks, either in the range of the movement or in the ease with which they can be elicited, is also strongly suggestive of disease.

When feeble the jerk can be reinforced or augmented by various means, as by asking the patient to grasp firmly, or to intertwine his fingers and then try to pull his hands apart as strongly as possible (*Jendrassik's method*). But the most efficient mode of reinforcement is to make the patient flex his great toe against some resistance, and to tap the tendon as he does so. If sitting, he may press his toe against the observer's shoe, or, if lying, against the bottom of the bed or the fingers of the ob-

server's left hand. If the knee-jerk cannot be then obtained it may be safely regarded as absent.

Though absence or diminution of the knee-jerk is evidence of disease, its increase or exaggeration is not necessarily so. It is true that the jerks are usually brisk or exaggerated in all cases of spastic palsy, but they may also be so in hysterical or nervous states, after exposure to cold, or when the patient's attention is directed to the test.

*Patellar clonus* can be frequently obtained when the jerk is increased by seizing the patella between a finger and thumb and pulling it suddenly downwards, or by striking a finger laid on its upper margin.

The **ankle-jerk** is obtained by tapping the tendo Achillis when the knee is slightly bent and the calf muscles are put on tension by dorsiflexing the foot passively; or the tendon may be percussed as the patient kneels on a chair with his feet over the edge. If at all brisk this reflex can also be elicited by a firm, sharp tap on the middle of the sole; this stimulus acts in the same way, that is, by suddenly increasing the tension in the calf muscles.

The ankle-jerks are constantly present in health, except perhaps in old age, and loss of them always indicates organic disease; they have, in fact, the same significance as the knee-jerks. When they are exaggerated a tap on the tendon frequently elicits a series of jerks if the foot is passively dorsiflexed by gentle pressure on the sole. Such a series, which constitutes *ankle-clonus*, can be brought out by dorsiflexing the foot abruptly and maintaining pressure on the sole. True ankle-clonus is found in all spastic conditions. A more or less similar phenomenon may occur in hysterical states, but then the jerks are less regular, do not persist so long, and are, in fact, merely a tremor due to the alternate contractions of the flexors and extensors of the ankle.

The tendon-jerks of the upper limb that are most commonly employed in clinical work are the extensor and flexor jerks of the **elbow**. The *extensor jerk* is obtained by striking the tendon of the triceps with a percussion hammer immediately above its insertion into the olecranon as the elbow is flexed to a right angle; this causes a contraction of the triceps which extends the elbow. Its spinal centre is in the seventh cervical segment. The *flexor jerk* is best obtained by pronating the arm, flexing the elbow to a right angle, and then striking the lower end of the radius briskly; this elicits

## REFLEXES

a reflex contraction of all the flexors of the elbow—biceps, brachialis anticus, and supinator longus—and, if exaggerated, a flexion of the fingers too. The spinal centre of the flexor jerk lies in the fifth cervical segment. A *pronator jerk* can be excited by percussing the posterior surface of the head of the radius as the arm is in the same position. Its centre also is in the fifth cervical segment.

All these reflexes have the same significance as the knee-jerk, that is, they are constantly present in health, exaggerated in spastic conditions, and diminished or lost when any part of their arcs is affected by disease or functionally depressed.

The **jaw-jerk** is obtained by percussing a finger placed on the chin as the patient allows his jaw to drop; when it is present the jaw is closed by a brisk contraction of the masseter and temporal muscles. It is exaggerated in bilateral spastic conditions and absent when the trigeminal nerves are affected.

**Superficial reflexes.**—The most important of these is the **plantar reflex**, which is obtained by stimulating the sole of the foot. The limb should be relaxed and supported, and stimulation is best done by drawing a pencil or a blunt object firmly along the outer border of the sole from the heel towards the toes, or by a pin if it is difficult to obtain a response. Stroking the soles normally excites reflex flexion and adduction of all the toes; this is the normal flexor response. But if the pyramidal tracts are affected by disease, or their functions temporarily depressed, as they may be after an epileptic seizure, the same stimulus elicits a slow extension of the great toe with spreading or abduction of the other toes, and in addition contraction of some of the thigh muscles, especially of the tensor fasciæ and the hamstrings. This is the *extensor response*, or *Babinski's sign*. The essential part of the reflex is the extension of the great toe at its metatarso-phalangeal joint; and thus, in the absence of local disease as paralysis or atrophy of the flexors of the toes, invariably indicates an affection of the pyramidal or cortico-spinal system. In infancy the normal response is extension.

*Oppenheim's sign* is closely allied to Babinski's, but it is neither so constant nor so reliable. When a blunt object or a finger is drawn firmly downwards along the inner border of the tibia the toes generally flex, but if the pyramidal tracts are affected this stimulation produces a strong dorsiflexion of the foot and extension of

## REFRACTION, ERRORS OF

the great toe, owing to contraction of the tibialis anticus and the extensor longus hallucis.

The **cremasteric reflex** consists in a drawing up of the testicle on the same side on stroking the inner surface of the thigh. It is usually absent in spastic paralysis of the same side, or when a lesion involves its centre in the first lumbar segment, but as it is inconstant and easily influenced by local conditions, as hydrocele, etc., its clinical importance is slight.

The **abdominal reflexes**, on the other hand, are of the greatest clinical value. Drawing a sharp or a blunt object over the lower margins of the ribs normally excites a contraction of muscles which pull the upper part of the raphé or the middle line of the abdomen towards the stimulus—the *upper abdominal* or *epigastric reflex*; a similar stimulus over Poupart's ligament or in the groins produces a displacement of the lower part of the raphé and the umbilicus towards the same side—the *lower abdominal* or *hypogastric reflex*.

These reflexes disappear when any disease affects the upper motor neurones; their absence is often the first indication of such an affection, preceding even the appearance of Babinski's sign. But it must be remembered that they may be abolished by many local conditions, as intra-abdominal disease or luxity of the abdominal walls, and obesity may make it impossible to observe them. And on the other hand they occasionally persist even in the presence of spinal disease, especially in children. Loss of those on the one side, or of the upper or lower only, in the absence of local disease is, however, always an important clinical sign.

The **corneal reflex** is elicited by touching the cornea gently with a blunt object, or by blowing on the eye; the stimulus produces a brisk winking action owing to contraction of the orbicularis palpebrarum. It is abolished when either the facial or the trigeminal nerve is injured. The **palatal reflex** is evoked by touching with a blunt object the margin of the palate on each side of the uvula; this produces an elevation of the arch of the palate on the same side. GORDON HOLMES.

**REFRACTION AND ACCOMMODATION, ERRORS OF.**—This article is intended mainly to assist those practitioners who add to their activities the treating of errors of refraction and the ordering of glasses. To those who have neither the time nor the inclination to undertake the correction of

## REFRACTION AND ACCOMMODATION, ERRORS OF

refractive errors, it may be pointed out that every practitioner must be familiar at any rate with its fundamental principles, since the cause of any defect of vision cannot properly be investigated without this knowledge. From a diagnostic point of view, he will save himself a good deal of anxiety if he can detect the movement of the shadows in hypermetropia, myopia, and astigmatism, and is able to estimate approximately the glasses which will correct the error. It is not at all uncommon for a patient to come complaining of defective vision in one eye which he has only recently discovered, and he therefore thinks he has suddenly lost his sight; this may or may not be a fact, as it frequently happens that a patient has had a defective eye for years without his knowledge until his attention was drawn to it accidentally. In such cases, one may be in doubt whether the cause is merely an error of refraction or is due to some intra-ocular disease. An acquaintance with the principles of retinoscopy will soon clear up this doubt, without being strictly accurate in its results, and, if the vision is materially improved with a correcting glass, an apparently serious case will soon appear far less formidable. Therefore, in cases of defective sight for which there is no obvious cause we should never miss the opportunity of estimating roughly the refraction, even though an operation may have been previously performed on the eye.

### ERRORS OF REFRACTION

I must recall a few elementary facts in connexion with the refraction of the eye.

Parallel rays of light, which for optical purposes means rays of light from an object at a distance of 6 metres and beyond, are focused on the retina of the normal eye, provided they do not subtend an angle of less than 5', *without any effort whatever*, and therefore distant objects are seen clearly with the eye at rest. Near objects emit divergent rays which can only be brought to a focus by increasing the refraction of these rays before they reach the retina. This is accomplished by an effort of accommodation whereby the curvature of the lens is increased, from relaxation of the suspensory ligament by means of the action of the ciliary muscles, thus augmenting its refractive power.

Any departure from the normal refraction of the eye constitutes an error of refraction, and the commonest of these are *hypermetropia*, *myopia*, and *astigmatism*.

In *hypermetropia* the eyeball is too short in its antero-posterior diameter, and is also often altogether smaller than normal. In such eyes parallel rays of light cannot be brought to a focus on the retina, but tend to be focused behind it. By an effort of accommodation of the requisite amount these rays can be focused on the retina, and therefore hypermetropic eyes are never at rest unless they are shut. A person with normal sight (*emmetropia*) and a hypermetrope of not too high a degree will see a distant object with equal clearness, and the latter may then be mistaken for normal unless special tests are applied to discover the difference. Hypermetropia does *not* mean the capacity for seeing distant objects more distinctly than a person can with normal sight; this is entirely a matter of cerebral observation.

In *myopia* the eyeball is longer in its antero-posterior diameter than normal, and therefore parallel rays of light never reach the retina, but come to a focus in front of it and after crossing at this point pass on, forming a blurred image of an object on the retina. Such eyes are adapted for the reception of divergent rays, which only occur when rays of light emanate from a near object. There is no mechanism in the eye capable of rendering divergent rays of light parallel, and therefore myopes never see clearly in the distance without the aid of glasses, whereas they have no difficulty in seeing near objects, and can often do so at a convenient distance without any effort of accommodation. Whilst hypermetropes are using their accommodation all day long and can see nothing clearly without it, myopes use their accommodation less than either emmetropes or hypermetropes, and sometimes hardly at all.

*Astigmatism* is a condition in which there is inequality in the radii of curvature of the various meridians of the cornea; this defect generally resolves itself into a difference between two principal meridians, which are always at right angles to each other, though not necessarily vertical and horizontal. Irregular astigmatism is where many of the meridians have different radii of curvature, and such cases can never be properly corrected. Lenticular astigmatism does exist in a certain number of cases, but the cornea is chiefly responsible for astigmatic errors. Astigmatism may be hypermetropic, myopic, or mixed (e.g. myopic in one meridian and hypermetropic in the other).

Convex glasses are used for hypermetropia,

## REFRACTION AND ACCOMMODATION, ERRORS OF

concave for myopia, and cylinders for astigmatism.

Hypermetropia and astigmatism are congenital in origin, and as a rule are not influenced by any strain of the intra- or extraocular muscles; nearly all infants are born hypermetropic, but gradually grow to a condition of emmetropia by lengthening of the eyeball. Myopia is seldom congenital, though occasionally such cases are met with, but, on the other hand, normal or even hypermetropic eyes may develop myopia as a result of eyestrain and muscular pressure. Heredity plays an important part in the development of myopia. This error is a common sequela of the corneal opacities which follow all forms of keratitis, and is aggravated by living under bad hygienic conditions. It is also found in commencing cataract when the lens is swollen, in cases of cyclitis, of conical cornea, and in subjects suffering from diabetes.

**Symptomatology.**—The symptoms common to all errors of refraction are blurring of the sight for distant or near objects, or both, and frequently in addition some headache or eyeache, due to overaction of the muscles of accommodation or of the extraocular muscles, alternating with fatigue of the muscle or muscles concerned due to their efforts to adjust the various defects; this constitutes the condition known as eyestrain.

Not all errors of refraction give rise to symptoms, and many people have quite marked defects without experiencing any discomfort whatever, so long as the vision remains fairly good; while, on the other hand, the smallest errors are said to be responsible for the most complicated train of symptoms, many of which appear to have nothing at all to do with the eyes. The fact remains that the symptoms are frequently out of all proportion to the degree of refractive error, and there is no constant relation between the two.

In *hypermetropia* there may be no symptoms whatever if the amount is only small, especially in young people, who are well able to carry off the increased accommodative effort necessary for accurate vision without feeling any discomfort; but, theoretically, blurring of the image should be noticeable for both distant and near vision. It is only when there has been some severe illness, followed by general muscular weakness, or after the age of 45, or from continual application to near work, that the ciliary muscle is unable to sustain the constant action necessary for maintaining normal

visual acuity; the patient is then conscious of indistinct vision and suffers from eyestrain, or is always rubbing his eyes under the impression that there is some film over the cornea which needs to be removed. These symptoms are naturally most pronounced after near work.

In *myopia* there are usually no symptoms of eyestrain, since the muscles concerned in accommodation are not much used; but spasm of accommodation does sometimes occur, when headache and eyeache may be complained of.

As a rule, the only symptom is defective distant vision, though short-sighted people may not always complain of it, and indeed often confidently assert that they can see perfectly well in the distance—a statement which may easily mislead the practitioner. Since myopia develops early in life, myopic patients do not always appreciate what clear distant vision means; they often say that they would rather not see any better, and complain of the vision being too brilliant and clear when correcting glasses are ordered. This fact must be remembered when prescribing glasses, otherwise the patients may be uncomfortable and refuse to wear them.

Another symptom is that all near work, i.e. books and needlework, is held very close to the eyes. This is not always an infallible sign of myopia unless the near point is excessively close, since all children, even the normal-sighted, hold their books closer than is absolutely necessary, in order to obtain a larger image, which is quite accurately focused even at this distance because of the high amplitude of accommodation common to all young people. Myopes, however, cannot read in any other way, and therefore holding the book close, in addition to defective distant vision, is practically conclusive.

In *astigmatism* headaches and eyeache are very common, and even slight errors give rise to severe and worrying symptoms, while the vision is also generally below the normal. There is much more strain on the ocular muscles in astigmatism than in either hypermetropia or myopia, but there is no special symptom to distinguish astigmatism from any other form of ametropia.

Conjunctival injection hardly amounting to definite inflammation, ciliary blepharitis, and other minor irritations about the lid are often aggravated or even caused by the eyestrain due to errors of refraction, and will not yield

## REFRACTION AND ACCOMMODATION, ERRORS OF

to ordinary remedies until correcting glasses are worn regularly.

The **diagnosis** of refractive errors is made by subjective testing or by retinoscopy, and usually both methods are employed. Retinoscopy is by far the more reliable of the two ; but the former is by no means to be despised even by those thoroughly experienced in refraction work. It often happens that the patient will not be comfortable with glasses merely indicated by mathematical calculation, and an intelligent and careful use of the subjective method, alone or combined with retinoscopy, is extremely valuable in deciding ultimately what glass may be worn with comfort.

**Retinoscopy or shadow test.**—The principle of retinoscopy is that rays of light reflected into an eye from a mirror will emerge from that eye according to its refraction. In an emmetrope they will emerge parallel, in a hypermetrope divergent, and in a myope convergent. A plane mirror (1 cm. diameter with a 3-mm. sight-hole) or a concave mirror (25 cm. or 10 in. focus, 3½ cm. diameter, with a 3-mm. sight-hole) may be employed ; I strongly advise the plane mirror, as with it the " shadows " are more easily detected and less experience is required.

It is advisable to have a rather larger sight-hole than is usually found in the ophthalmoscope, and therefore special retinoscopy mirrors are sold for the purpose, but with a reasonable degree of experience the ophthalmoscope can generally be used.

The examination can be conducted at a considerable distance from the patient, and, speaking generally, the shadows can be more easily detected the farther off the surgeon stands ; as a rule, however, it is found most convenient to sit opposite the patient one metre away, so that the glass can be readily adjusted in the trial frame without unnecessary movement. Retinoscopy can be carried out either with or without a mydriatic, but if there is any difficulty experienced with an undilated pupil it is better to instil atropine or homatropine. In children, who generally find difficulty in relaxing their accommodation while under examination, it is better to employ a mydriatic, and in them atropine ointment is prescribed, to be put into the eyes three times a day for a week ; but adult patients, by looking beyond the surgeon's head into the distance, are able in many cases to produce sufficient relaxation to make the refraction comparatively easy. In all cases in which the

vision cannot be improved to  $\frac{2}{3}$  a mydriatic must be used, provided there is no indication against it, since, apart from the refractive error, some disease of the eye may coexist to account for the lowered standard of vision. Homatropine is the mydriatic suitable for adults, as it quickly paralyses the accommodation and the effect only lasts for about thirty-six hours.

The common teaching is that it is dangerous to put atropine or homatropine into the eyes of patients over 45, and this often leads the practitioner to withhold the drug in cases where it is absolutely necessary to use it. Only in those who show certain tendencies to increased tension is it necessary to be careful, or to avoid using a mydriatic altogether. The contraindications are a shallow anterior chamber and a tendency to a dilated pupil and increased tension. If the practitioner is in doubt, an inspection of the disc before application will in most cases solve it.

Take the patient into the dark room and let him be seated on a chair with his back to the light, which must be arranged just above his head a little behind the level of the forehead so that the eyes are in the shade. Sit down exactly opposite to him at a distance of one metre. If he is under a mydriatic, direct him to look at your forehead just above the right eye, and examine each eye separately in the way now to be described, using a trial frame in which a block is placed in front of the eye not under examination. The practitioner must be careful to put up his own correction (if any) behind the sight-hole in the retinoscope.

Reflect the light into the patient's eye with the mirror close up to your own eye, so that the latter is just behind the sight-hole. The first thing that is seen is the red reflex from the fundus illuminating the pupil. This illumination will be bright or dull according to the refraction of the eye under observation, the lower errors of refraction being associated with a bright and the higher with a dull illumination.

In order to see the shadow, move the mirror, usually by tilting it, so that the illuminated area moves slowly across the pupil. It is important to remember that, until one is thoroughly experienced, these movements should be slow and deliberate ; rapid and wide movements make it very difficult, if not impossible, to see the shadows distinctly. The actual shadow is merely the line of contrast between the light and the dark part of the reflex, and

## REFRACTION AND ACCOMMODATION, ERRORS OF

the fewer the rays of light which reach the retina the darker will be the whole reflex, and therefore the darker the shadow. The shadow is dark in high errors of refraction, whether hypermetropic or myopic, or with some opacity in one of the media, either cornea, lens, or vitreous, which also causes a dark reflex. Carry the light slowly across the pupil, first in the horizontal and then in the vertical meridian, and note the way in which the shadow moves. If it follows the *same direction* as that of the mirror (plane) the correcting glass will be a convex (or  $+$ ) one, and the error will be either hypermetropia or low myopia, or the eye may be emmetropic (normal); if the movement is *against* the mirror the error will be myopia of a moderate or extreme degree and the correcting glass will be a concave (or  $-$ ) one.

Sometimes the shadow moves in the same direction in one meridian and in the opposite direction in the other meridian; in this case we are dealing with mixed astigmatism. With the same type of glass one meridian may be corrected with a higher strength than the other; this indicates simple astigmatism, either hypermetropic or myopic, and a note must be made of the correcting glass for each meridian. As a rule there are two principal meridians only, at right angles to each other, though irregular astigmatism occasionally occurs, giving rise to the so-called scissor movements, by which is meant shadows moving in all directions across the pupil. This cannot accurately be corrected by any glass, and only an approximate calculation can be made of the refractive error in these circumstances. Inasmuch as there is a certain amount of spherical aberration in most lenses, we must learn to pay attention to the central part of the reflex rather than the peripheral, for it is through this part that the patient will see when the pupil becomes the normal size after the effect of the mydriatic has passed off.

The object is to find the point of reversal with the correcting glass, and this will be the case when the rays of light come to a focus on the pupil (or, more strictly speaking, on the nodal point of the eye) of the observer; the illumination is then at the height of brilliancy, and therefore no shadow is seen at all.

Parallel rays are brought to a focus at a distance of one metre by a  $+1$  D spherical lens. Since the rays emerging from an emmetropic eye are parallel, and one metre is the distance at which the patient is sitting, his

eye is emmetropic if the point of reversal is secured by a  $+1$  D spherical lens. Correcting glasses must, therefore, be calculated on this basis.

The rays of light from a hypermetropic eye emerge divergent, and, by introducing  $+$  or convex lenses into the trial frames, are gradually brought down to parallelism, when an extra  $+1$  D must be added in order to focus them on the nodal point of the observer's eye; therefore, in calculating the correcting glass for the patient this amount must be deducted. Similarly, from the myopic eye the rays of light converge, and by means of  $-$  or concave lenses are brought to a position of parallelism; but before they become parallel they converge on to the nodal point of the observer's eye, i.e. the point of reversal of the shadows has been reached; therefore, as we desire to make the rays parallel for the patient, we add 1 D on to this correction to arrive at the actual amount of myopia.

From this we evolve the following rule: Deduct 1 D from all  $+$  corrections, and add 1 D to every  $-$  correction. If the correcting glass is less than  $+1$  D, e.g.  $+0.5$ , then by deducting 1 from this the correcting glass is found to be  $-0.5$  D; thus low degrees of myopia show the shadow moving in the same direction as the mirror, and are corrected by a convex lens.

The practitioner must keep clearly in his mind the meaning of this calculation: it has to be made because it is more convenient to sit at a distance of one metre; if he chooses to stand two metres away, then 0.5 D must be deducted or added, as the case may be; if it were possible to do a refraction at six metres, no deduction at all would have to be made. Other calculations which have to be made from the correcting glass measured in the dark room are dependent on the tone of the ciliary muscle, which varies in different subjects, but this particular one remains constant because it is attained mathematically and has nothing to do with the physiological functions of vision.

*Variations in the shadows.*—It is comparatively simple, after a little practice, to see the shadows in simple hypermetropia, in myopia of a not very high degree, and also in emmetropia, but there are certain variations in its appearance which require some explanation, and which may cause the practitioner to doubt his powers of observation.

I have already alluded to one, viz. the scissor



## REFRACTION AND ACCOMMODATION, ERRORS OF

movement in cases of irregular astigmatism. Another is the band-like shadow seen in advanced astigmatism, where the edge of the shadow forms a straight line across the papillary area; the direction of this linear edge indicates the axis of the astigmatism. If the astigmatism is of high degree this band is noticeable before any spherical glass is placed in front of the eye, but if of low degree it does not appear until after the spherical correction has been made.

A brilliant illumination with hardly any shadow to be seen, or at any rate with some difficulty in appreciating its direction, means a low error of refraction, for this shows that we are already near the point of reversal; the same appearance is seen in moderate degree when the correcting glass has been nearly reached. A very dull illumination, with a similar difficulty in appreciating the shadow and its direction, may indicate, as previously mentioned, a very high error of refraction, either myopic or hypermetropic; or, again, it may mean that there is some opacity in the cornea, lens, or vitreous which is obstructing the rays of light. The best way of settling this question is to try to decide which way the shadow appears to go and then introduce at once a high spherical lens of the proper sign (+ or -) into the trial frame. For instance, if we think the shadow is moving in the opposite direction to the mirror, we put a high concave glass in front of the eye, e.g. a - 8 sphere. If now the illumination is more brilliant than before and we see the shadow more distinctly, yet the movement is still against the mirror, we know that the correcting glass has not yet been reached, and we must go on putting up higher concave lenses. If, however, this glass makes the illumination duller than before, we know that this cannot be the right kind of glass, and we then introduce a high convex glass, e.g. a + 8 sphere. If now the shadow becomes clearer, the illumination brighter, and the shadow moves in the same direction as the mirror, it follows that the correction required is hypermetropic, and we proceed to introduce higher convex lenses till we reach the point of reversal.

If a concave mirror is used for retinoscopy the shadows behave exactly in the opposite way to that described for the plane mirror.

**Subjective testing** is very convenient, and with an intelligent and observant patient may prove of great value, whether by itself or as an adjunct to retinoscopy.

If carried out independently of the shadow test, it may be undertaken in the following way: Let the patient be seated six metres away from a Snellen's test-type board (or at five metres with test type arranged accordingly), and place the trial frame on the face adjusted as near the eye as possible and with the patient's eye looking straight through the centre; test each eye separately by carefully blocking out the view of the eye not under examination.

Ask the patient to read the letters of the Snellen's test types and notice the lowest line he can read correctly. If he reads the last line correctly he is either emmetropic or hypermetropic; he cannot very well be myopic, though he may be slightly astigmatic. To differentiate between the first two, place a small convex spherical lens of  $\frac{1}{2}$  D in front of the eye in the trial frames, and ascertain whether he can still read the letters on the last line correctly: if he cannot, he is emmetropic; if he can, he has some hypermetropia. Increase the strength of the convex glass until he states that the last line has become blurred; the glass just below this in strength represents the amount of his hypermetropia and is called the *hypermetropia manifest* (H.M.). If the patient is young there will in all probability be a further amount of hypermetropia, which can only be determined after paralysing the accommodation with a mydriatic, and is at present hidden owing to the effect of the tone of the ciliary muscle. This is called the *latent hypermetropia*, and must be allowed for if symptoms are to be relieved. If the patient is older, and especially if he is nearing the age of 45, the full amount of hypermetropia may be arrived at by the subjective method alone.

If the patient is unable to read the last line on the board, but stops short of it by one or two lines or more (e.g.  $\frac{1}{2}$ ), then put up small convex spherical glasses and see if improvement is possible, and how far this improvement can be carried. If by this method two lines farther down can be read (=  $\frac{2}{3}$ ), but no farther, in spite of increasing the strength of the glass, there is probably some astigmatism in addition to the hypermetropia. Then replace the glass which enabled the patient to read  $\frac{1}{2}$  and add small convex cylinders, beginning with + 0.5 D, or even lower, with the axis vertical; if this does not bring about any improvement, place the same cylinder with its axis horizontal; if the patient is still unable to read any farther,

## REFRACTION AND ACCOMMODATION, ERRORS OF

rotate the cylinder into various diagonal meridians and see if any other position, besides the horizontal or vertical axes, is appreciated. By this means the correct axis will soon be arrived at. Having once determined this, increase the strength of the cylinder until the patient reads  $\frac{5}{6}$ .

If neither convex spherical nor cylindrical glasses improve the vision, try small concave spherical lenses and cylinders in the same way as in hypermetropia, but place the cylinder first in the horizontal meridian before trying any others, always bearing in mind that subjective testing with concave glasses is never so reliable as with convex, and that a sharp lookout must be kept when the point of correction is reached and the patient reads  $\frac{5}{6}$ . It is easy to over-correct myopes, as they can see equally well with a rather higher glass than full correction; but if the rules to be laid down later are carefully followed there need be no fear of this in adults.

In young people it is seldom wise to rely on subjective testing in myopia, but the refraction should nearly always be done under a mydriatic.

The guide to over-correction in hypermetropia is blurring of the letters (of  $\frac{5}{6}$ ), and in myopia a diminution in the size of the letters, though the patient may give no indication of this unless asked the question.

In both hypermetropic and myopic astigmatism, unless the spherical glasses materially improve the vision, discard them and begin at once with a cylinder.

A good method of verifying the axis of the cylinder is by means of the astigmatic fan, which may be utilized in two ways. One way is to show the patient the fan with the glasses on, and ask if all the spokes are equally distinct; then remove the cylinder or place it in a different axis, and ask him again if all the lines are distinct: an intelligent patient will generally be able to appreciate accurately the difference. The other way is to put up the correction and get the patient to read  $\frac{5}{6}$ , then put an additional + 3 D on the spherical correction and omit the cylinder; this fogs all the letters. After a wait of a few minutes the ciliary muscle completely relaxes; then add minus spheres until some of the lines on the astigmatic fan appear clear while those at right angles to them are still foggy. Now add minus cylinders with the axis at right angles to the blurred lines, and, as soon as the lines are all manifestly black and well

defined, get the patient to look again at the letters and see that they are all distinct.

E.g. The correction is

+ 1 sphere

+ 2 cylinder axis vertical

Put up + 5 sphere, and by gradually adding minus spheres and minus cylinders get the patient to see all the spokes of the fan clearly. If in order to do this a - 2 sphere and a - 2 cylinder at horizontal axis must be used, this

+ 1 sphere

makes the correction + 2 cylinder axis vertical.

If by any or all of these methods the practitioner is enabled to improve the vision of a patient whose visual acuity is far below the standard, even though he may not be able to obtain perfect vision in each eye, a distinct advance has been made towards the diagnosis.

The subjective method is also applied *immediately after estimating the correction by retinoscopy in the dark room*; in this case the practitioner may put up the glasses he has arrived at and from past experience make the necessary calculations for the return of the tone of the ciliary muscle after the effect of the mydriatic has passed off, or may carry out a post-mydriatic subjective test, whichever he prefers.

Up to this point, both the practitioner who only wishes to make a diagnosis and one who desires to go a step further and order glasses must proceed in the same way.

**Treatment.**—The usual rule when prescribing glasses is to give the lowest in myopia which the patient will accept binocularly for distance or constant wear, and the highest in hypermetropia for constant wear; while in astigmatism the cylinder must not be altered. An exception is when the cylinder is one of high power and is to be worn for the first time in adult life, in which case the lowest cylinder (either hypermetropic or myopic) with which the patient can read the lowest line may be ordered. This is an excellent working rule; there is little fear of over-correcting if it is strictly followed, and patients will generally be comfortable. Only occasionally will it be necessary to order any special reading-glasses unless the patients are over 45.

The prescribing of glasses must be carried out in the following way:—

The correcting glasses having been determined by retinoscopy, the patient stands six metres from Snellen's test types, and glasses of the required strength are placed in the trial frame, after deduction or addition, as the case

## REFRACTION AND ACCOMMODATION, ERRORS OF

may be, has been made for the distance at which the practitioner sits when conducting the refraction in the dark room. As this distance is usually one metre, the amount to be added or subtracted will be 1 D.

For example, if the case is one of hypermetropia, when the correcting glass in the dark room was 3 D we put up a + 2 D in the trial frames and the patient ought to read  $\frac{5}{6}$ . Similarly, in a case of myopia in which the correcting glass in the dark room was - 3 D we put up a - 4 D, when the patient should read  $\frac{5}{6}$ . These, therefore, will be the glasses respectively which a patient will require in order to read  $\frac{5}{6}$  when under a mydriatic. But when the effect of the cycloplegic passes off, the tone of the ciliary muscle returns: this keeps up, at any rate for a time, a certain amount of accommodation without any definite muscular effort, and this same tone has to be reckoned with in patients whose refraction is undertaken without mydriasis.

Since this is a purely physiological process, it varies in different persons and in the different forms of refraction, and therefore any calculation made on this account cannot follow a rigid mathematical rule, as can that made for the distance at which the practitioner sits in the dark room.

In young people this tone is likely to be well marked in any case, and in hypermetropic patients who have used their ciliary muscles excessively owing to the necessity for continual accommodation, for distance as well as near, it will be well developed, and the more so the higher the error; whereas in myopia, where accommodation is not even so necessary as in emmetropia, it is likely to be weaker than normal, and in very high myopia may indeed be in an atrophic condition. As a matter of fact, approximate calculations can be made in most cases if we bear in mind the above reasons for variations, and suitable glasses can be ordered immediately after the subjective test following the retinoscopy; but if the practitioner is in any doubt, the best plan is to get the patient to return in a fortnight's time if atropine has been used, or in three or four days if homatropine has been used, and then endeavour to get him to take the highest convex glass in cases of hypermetropia which he will accept binocularly, as near the retinoscopy correction as possible. It will generally be found that this amount falls short of the retinoscopy correction by about 1 D, but it may be more in young people

and in cases of high hypermetropia. After the glasses have been worn for about six months, it is often found possible to substitute a glass slightly higher and nearer the full correction; this means that the tone of the ciliary muscle is being overcome, and this extra strength should be added to the glasses and new ones prescribed accordingly.

In myopia, on the contrary, it will be found that practically no calculation need be made for the tone of the ciliary muscle, and the glasses with which the patient is most comfortable are those actually made out by retinoscopy after the addition of the usual 1 D for the distance at which the practitioner sits in the dark room. Perhaps *binocularly* the patient may prefer even 0.5 D less than this; if so, these are the glasses which should be ordered. In high degrees of myopia patients are often much more comfortable with 3 D or 4 D less than full correction, in spite of the fact that some of their distant vision has to be sacrificed; they complain that the full correction is much too brilliant and "dazzles" them.

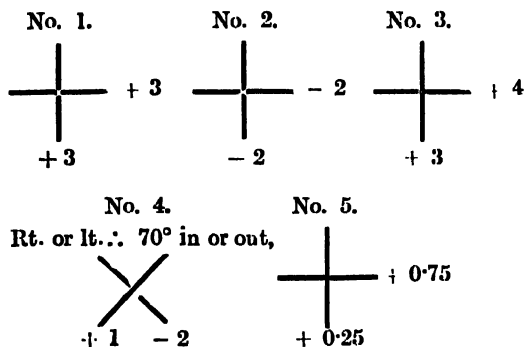
The above alterations apply *only* to spherical lenses. Astigmatic corrections in every case are made to counteract refractive errors due to the abnormal curvature of the cornea, and are independent of the tone of the ciliary muscle; therefore, theoretically, no alteration in cylinders should be made. As a matter of practice, if the cylinder is a high one, and in many cases of mixed astigmatism, a somewhat lower correction than the actual mathematical figure will be accepted more readily without affecting the visual acuity. In mixed astigmatism this is accounted for by the fact that it is very easy to over-estimate the strength of the cylinder. For example, if one meridian is corrected by a + 1 D and the other by a - 1 D, it is often difficult for even experienced refractionists to be sure of the reversal of the shadow with a + 1 D or + 1.25 D, or with a - 1 D or - 1.25 D; this does not matter in simple astigmatism, where the signs (+ or -) are the same, since both meridians will be over-corrected and the subtraction would be the same with the higher as with the lower correction; but in mixed astigmatism the difference would be + or - 2 D as compared with + or - 2.5 D, so that an over-estimation to the extent of only 0.25 in each meridian makes a difference of 0.5 D in the cylinder.

Every care must be exercised in estimating accurately, both by retinoscopy and by sub-

## REFRACTION AND ACCOMMODATION, ERRORS OF

jective testing, the axis of the cylinder, particularly if it be a high one—for there is no point upon which a patient is more sensitive than this, and a cylinder placed in the wrong axis may actually give rise to eyestrain, and is often the reason why glasses are not comfortable or fail to relieve the symptoms.

*Examples (under mydriatic):*



No. 1: +2 sphere to be ordered eventually, but only +1 D accepted at first.

No. 2: -3 sphere to be ordered, and probably accepted at once, or 2.75 binocularly.

No. 3: +2 sphere to be ordered eventually, but perhaps only +1 sphere accepted at first.  
+1 cylinder axis vertical

No. 4: Two forms of glass. Either -3 cylinder axis 70° down and in, or down and out; or -3 sphere  
+3 cylinder axis 20° down and out, or down and in.

No. 5: -0.25 sphere to be ordered, and probably accepted at once.  
-0.5 cylinder axis horizontal

Where the difference in the strength of the glasses prescribed for each eye is very great, as with a -8 D in one eye and a +2 D in the other, such glasses will never be worn, although the vision in each eye with correction may be  $\frac{5}{6}$ . The images perceived by each eye in such cases are not the same size, and so cause confusion. This is known as *anisometropia*, and in spite of the good vision one eye must be neglected and monocular vision alone obtained with the correcting glass, but the actual amount of anisometropia which can be tolerated varies very much, and, unless there is a gross difference between the two, full correction should be tried. Other errors of refraction are, where

the lens has been extracted for cataract, when a high convex glass, about +12 D, generally combined with a cylinder in the horizontal axis, is necessary for correction; and also in cases of conical cornea. This latter instance is rare, and the usual correcting glass is a very high cylinder, generally concave, with its axis vertical, often combined with an equally high sphere; but in both these cases subjective testing has often to be relied upon, as retinoscopy is difficult in the former and impossible in conical cornea. After cataract extraction with a good hole in the capsule, an accurate refraction can generally be managed.

The advice to be given in each case, when glasses are prescribed, is a matter of importance, as most patients regard the wearing of glasses as a serious handicap in life, for various reasons, and many are anxious to avoid wearing them if possible. The practitioner must therefore be very clear in his own mind as to their uses, or he will be over-persuaded.

In *hypermetropia*, the main symptoms being headache and eyache from over-use and fatigue of the ciliary muscle, the patient will be ready enough to take advantage of the relief that glasses afford, but in all cases the objection to constant wear is the main one. In young people, and in those whose error of refraction is not of a high degree, or whose symptoms are not constantly present, it is only necessary for the glasses to be worn for reading, writing, and sewing; if this does not relieve the symptoms of eyestrain, then they must be worn constantly. Older people, over 45, in whom accommodation is beginning to fail, will usually find it necessary to wear glasses for distance as well as reading, though if the vision for distance is not very far below normal, as in low degrees of hypermetropia, distance glasses can be dispensed with, since vision of  $\frac{1}{2}$ , for example, is quite good enough for all practical purposes in everyday life. If glasses for distance as well as reading are needed, a good way is to combine them in one glass as bifocal lenses. In these circumstances the patient must be advised, on going downstairs, to depress the head slightly so as to ensure the distance lens coming into use; otherwise he may stumble from looking through the reading half of the glasses. When there is present some chronic conjunctivitis or blepharitis which steadily defies all methods of treatment, it may be due to eyestrain; in this case, if correcting glasses for the error of refraction be worn constantly, the inflammatory

## REFRACTION AND ACCOMMODATION, ERRORS OF

symptoms will often clear up extraordinarily quickly.

Although patients are much distressed at suffering pain, and think that on this account there is danger of losing their sight, no secondary complications are likely to arise in hypermetropia, and they may be reassured on this point. They can be told that unless they have symptoms of headache, eyeache, etc., or blurring of the sight, they need not wear glasses except for reading.

It is in *myopia* that difficulty arises in giving advice. The general public regard short sight as an advantage rather than otherwise, and do not take it seriously enough. The common remark one hears is, "Then he will get better as he gets older," and as many patients do not seem to notice the disadvantage of indistinct distant vision, and can always read quite easily, they refuse to believe that any serious consequences are likely to follow from discarding their glasses. Since myopes generally have a fairly normal amount of accommodative power, except those with a very high degree of error, patients under 45 can read comfortably both with and without glasses; but when they reach that age the convex lens necessary for presbyopia is counteracted by part of their myopia, so that by doing a little less accommodation with their glasses off they can read perfectly well, though perhaps at somewhat closer range, and appear to be in exactly the same position as before; and so on up to quite advanced age, according to the degree of myopia. When they arrive at the age of 60 or 70, provided their myopia is above 4, they are still able to read without glasses. Comparing themselves with their normal presbyopic companions, who are wearing glasses, they consider themselves in a far superior position, and believe that their eyes are therefore very strong and have improved. This is the origin of the popular and fallacious notion that short-sighted people always get better as they get older; they fail to observe that their distant vision is the same as before.

In the present state of our knowledge we regard prolonged application to near work as a predisposing cause of myopia, given some inherent weakness of the sclerotic coat. Most young people hold books much closer to the eyes than is necessary; this compels them to keep the eyes fixed in one position for a considerable length of time, and calls into play a large number (perhaps all) of the extra-ocular muscles, since no movement of the eyes is

really a simple one. The pressure of these muscles, which are attached to the globe, in patients with weak sclerotics, will cause the eyeball to bulge at the only point where it is unsupported by muscles, viz. the posterior pole of the eye, and thus the eye becomes slowly longer and more myopic. This is not noticed if it is a gradual process, and the myopia becomes "progressive." Such increase in the myopia is likely to occur during the growing period of life, but in adult life, when the sclerotic becomes firmer, the myopia will remain stationary. Therefore the risk in early life is progressive myopia, which may lead to splitting of the choroid and retina in the macular region, with consequent defect in vision which can never be improved; detachment of the retina often occurs also.

Advice to myopic patients should be prophylactic: they should wear glasses constantly to encourage the eyes to behave more naturally, should never hold the book close to the eyes, never read very small print, and never read in a bad light. If, in spite of this discipline, the myopia progresses at all rapidly, then all near work must be stopped and attention paid to the general health by open-air life, with plenty of good food, tonics, etc.

A common question for the practitioner to be asked is whether the glasses may be left off for distance, or at any rate occasionally. If patients are willing to put up with the discomfort of imperfect distant vision, theoretically there is no objection to this; but unless they wear the glasses regularly they are almost sure to forget them for reading, as they experience no difficulty at that time.

### ERRORS OF ACCOMMODATION

The commonest error of accommodation is *presbyopia*, or so-called "old-age sight." This is not really a refractive error, but is merely the normal physiological failure in the amplitude of accommodation when the lens is no longer elastic but is undergoing the process of sclerosis of the nucleus, and therefore cannot respond to the contraction of the ciliary muscle. This change begins to show itself about the age of 45. It is corrected by convex lenses of suitable strength for a given age. The usual rule is as follows: +1 D at 45, +1.75 or +2 D at 50, +2.5 D at 55, +3 D at 60, +3.5 D at 65. But since presbyopia is of physiological origin, it must vary within certain limits, and no hard-and-fast rule can be laid down as to the strength of glass suit-

## REFRACTION, ERRORS OF

able for each case. The above figures must only be regarded as a basis on which to work. All presbyopes must first be tested for distance; the strength of glass is calculated from the age of the patient, due allowance being made for the correction which has been found necessary, either for myopia or hypermetropia, in order to procure perfect distant vision ("). Then test subjectively, and ascertain the *lowest* convex glass with which the patient can see most distinctly the smallest Jaeger type at eighteen inches, and it will generally be found that this works out considerably lower than the figures given above. Nothing worries patients so much as being forced to do all sorts of near work, writing as well as reading, at an inconveniently close range, and that is the reason for choosing eighteen inches' distance and the lowest convex glass.

Some patients prefer to have a stronger pair of glasses for reading and a weaker pair for writing or music, but it must be remembered that strong glasses necessitate close range, and, as weakness of convergence proceeds hand in hand with weakness of accommodation, the strain on the internal recti muscles must be relieved by the addition of prisms with bases inwards if close range and strong glasses are essential, as for some forms of near work.

*Paralysis of accommodation* occurs sometimes after diphtheria and in some forms of acute illness, and is only discovered by noticing that a patient can read " in each eye but is unable to read any but the large Jaeger types; such cases illustrate the importance of testing both distant and near vision in every case. As a rule this condition gradually improves, but if not, glasses for reading must be prescribed as for presbyopia.

*Spasm of accommodation* is met with in young hypermetropic patients, sometimes in myopia, and also in neurotic subjects. It produces an artificial myopia, and also headache and eye-ache; therefore patients will complain, no matter what glasses are worn, and show great variations in refractive error from time to time, which cause them to be continually changing their glasses.

The treatment is to correct the refractive error by careful retinoscopy under full mydriasis, and to prescribe prolonged rest from near work for a time if necessary, dark glasses, and attention to the general health. But these cases are often very troublesome, and baffle even the most experienced.

MALCOLM L. HEBURN.

## RELAPSING FEVER

**REGURGITATION** (*see* STOMACH, FUNCTIONAL DISORDERS OF).

**REICHMANN'S DISEASE** (*see* STOMACH, FUNCTIONAL DISORDERS OF).

**RELAPSING FEVER** (*syn.* Famine Fever, Remittent Fever, Spirillar or Spirochaetal Fever, Remittent Icteric Fever, Bilious Typhus Fever, Tick Fever, Relapsing Fever of Tropical Africa).—An acute specific fever, characterized clinically by recurrent attacks of fever lasting about a week, spaced by apyrexial periods of similar duration, and pathologically by the presence of a parasite in the circulating blood.

**Historical.**—The disease was separated, on clinical grounds, as a distinct entity by Henderson, of Edinburgh, in 1842, and the parasite which bears Obermeier's name was described by that worker in 1873. Previously the disease had been confounded with typhoid, and particularly with typhus fever, to which it is closely allied in epidemiology. It is now known to be a world-wide disease, though its course and the character of the spirochaetes vary slightly in different parts of the world.

**Etiology.**—Relapsing fever is intimately associated with poverty, overcrowding, starvation, and other concomitants of social degradation. The carrier was believed to be the bed-bug until Mackie in 1907, working in India, showed that this insect played no part in its transmission, but that the true intermediate host was the body-louse (*Pediculus vestimenti* or *corporis*). Subsequently, Sergeant, Nicolle and others confirmed and extended the louse hypothesis, which may be accepted as the correct one for most parts of the world. In tropical Africa, Arabia, and possibly in Persia, ticks of the genus *Ornithodoros* carry the disease. (PLATE 38, Figs. 4, 5, facing p. 366.)

Experimental evidence shows that the spirochaete may be absorbed by unbroken mucous membrane, from the conjunctival sac, through minute abrasions of the skin, and also by feeding with infected blood. Possibly these may supplement the ordinary method of transmission during epidemic times. The writer has also shown that an epidemic among the staff of a lying-in hospital was due to contact with the fresh placental blood of women who aborted during the course of relapsing fever.

**Pathology.**—The spirochaetes of relapsing fever found in different parts of the world have received specific names, according to differences in morphology, animal or serum reaction; e.g.

## RELAPSING FEVER

*S. recurrentis* (Lebert, 1874), or *S. obermeieri* (Cohn, 1875), in Europe (Fig. 85); *S. duttoni* (Novy and Knapp, 1906), in tropical Africa; *S. carteri* (Mackie, 1907), in Asia; *S. novyi* (Schellach, 1907), in America. Pathologists are not agreed whether these distinctions are specific.

The parasite appears in human blood within twenty-four hours of the onset, and multiplies until the crisis, when it is destroyed by the accumulation of spirillicidal substances in the serum, and is then phagocytosed in the internal organs.

The behaviour of the spirochæte in the insect vector is not fully understood. According

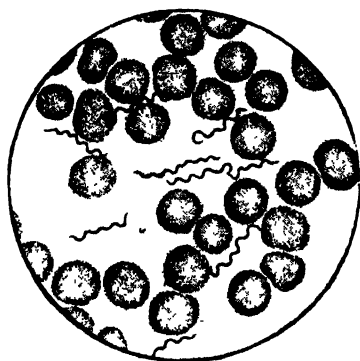


Fig. 85.—*Spironema recurrentis* in blood-film.  
× 500. (Microphoto: Dr. John Bell.)

to Nicolle and his co-workers, multiplication goes on in the body cavity of the louse, and infection occurs by the insect being crushed into the skin surface, abraded by scratching. Their experiments proved that the disease is not ordinarily transmitted by the louse in the act of biting. Leishman found that, in the tick, the spirochæte passed into a coccoid or granular stage and that the granules were transmitted to man during or after the act of biting. The granules found in the cold-blooded arthropod redeveloped into typical spirochætes in the warm-blooded host.

Post-mortem changes are not distinctive, but resemble those of any acute septicæmia. Areas of necrosis and hæmorrhages are found in the liver and spleen, especially in the bilious-typhus type of relapsing fever.

**Symptomatology.**—The incubation period varies between two and ten days, but is generally about a week. Invasion is rapid or sudden, with chills or rigors, headache, pains in the limbs, and high fever, reaching 104° or 105° F. at the first bound. The temperature then

drops slightly, but remains high with morning remissions and tends to become higher towards the crisis. Then it falls suddenly, often to 96° F. or 97° F., gradually rising to reach normal at the time the relapse is due.

**Alimentary system.**—Marked anorexia and bilious vomiting are almost invariable. The tongue is moist and furred at first, but in severe cases becomes dry, brown, and cracked. At first the bowels are confined, but diarrhœa is common in the later stages, and discharges of mucus and blood are not rare.

**Circulatory system.**—The pulse quickens with the fever and may attain great frequency—160 to the minute—towards the crisis. The heart is very susceptible to the toxin of relapsing fever, but organic changes are very rare. Persistent tachycardia, or, less commonly, bradycardia, is an occasional sequela. The blood shows a pronounced leucocytosis, chiefly due to increase in the polynuclear and lymphocytic elements. This leucocytosis drops at the crisis. Apart from the presence of the specific parasite, the blood changes are not diagnostic.

**Respiratory system.**—Epistaxis is common at the onset. Mild bronchitis is a frequent symptom, whilst lobular and croupous pneumonia are also met with. Scattered patches of consolidation in the lungs, probably due to hæmorrhagic infarctions, also occur, and are toxæmic in origin. Croupous pneumonia is caused by the pneumococcus, and not by the spirochætes.

**Nervous system.**—Intense headache is often complained of at the onset, and in some cases gives place to delirium, in others to somnolence or mental hebetude associated with the "typhoid state." Maniacal outbursts are sometimes met with during the height of the fever, or even in the apyrexial period. A meningeal type of the disease is recognized, but is rarely met with.

**Cutaneous system.**—Purpuric or petechial rashes are described. They are most common on the trunk and in hæmorrhagic and toxæmic cases. Not infrequently they are merely louse-bites.

**Urinary system.**—Bile-stained urine is almost invariable, and is certainly present in all severe cases. A cloud of albumin is demonstrable during the fever, but organic kidney disease does not follow. Hæmaturia is present in hæmorrhagic cases.

**Reproductive system.**—Women who are pregnant almost invariably abort, and the placental blood contains the spirochæte. Orchitis is an occasional complication.

## RELAPSING FEVER

**Special senses.**—Eye complications are especially liable to occur in African tick fever, but occasionally also in other types of the disease. The lesion is a keratitis or an iridocyclitis, which may destroy the whole eye.

**Relapses.**—These vary in number and intensity with the virulence of the epidemic and the resistance of the patient. The first apyrexial period is generally about seven days, but with each successive relapse the tendency is for the pyrexial period to get shorter and less severe, and for the subsequent afebrile period to lengthen. The liability to relapses varies greatly in different epidemics. In African tick fever they are often numerous, amounting even to eight or ten, and the duration of the disease consequently covers a period of many weeks. Generally they are relatively mild, but in very debilitated patients the first relapses may be more severe than the original attack, and death may ensue from exhaustion and cardiac failure.

**Special clinical types.**—**Ambulatory cases** may occur among contacts in an infected community. Although in such cases the patients may have only suffered from transitory fever, spirochaetes may be found in their blood. These cases are important, as the patients may act as "reservoirs" or "carriers" of the disease.

**The bilious-typhus type.**—This dangerous form of relapsing fever may occur along with the milder variety, and epidemics largely owe their relative mortality to its degree of frequency. Jaundice sets in early, and the abdomen is tumid, while the liver and spleen are enlarged and tender. The eyes are injected and deeply jaundiced, the tongue dry and the lips covered with sordes, while bilious vomiting is the rule. The disease runs an adynamic course, the temperature ranging lower and failing to come down at the crisis. The patient develops hiccough, tympanites, and low delirium, and passes into the "typhoid state." Death takes place usually from cardiac failure, the result of the profound toxæmia.

**Hæmorrhagic type.**—A tendency to bleeding often complicates the bilious-typhus type, but may arise independently. Prolonged epistaxis, hæmatemesis, purpuric skin eruptions, melæna and hæmaturia are all liable to occur.

**Diagnosis.**—The only certain method is to demonstrate the spirochaete in the peripheral blood. Clinically, it is likely to be confounded with typhus fever and the severer tropical fevers of short duration such as dengue, some-

## REMEDIAL EXERCISES

times with typhoid or yellow fever, pneumonia and septicæmic plague, and rarely with cerebro-spinal meningitis. The association of high fever, developing rapidly, with icteric conjunctivæ and bile-stained urine, is very suspicious. When relapses occur, the disease can hardly be confounded with any other.

Serum reactions are of diagnostic value, but not usually available to general practitioners.

**Prognosis** varies enormously in different epidemics and according to the environment. In clean and well-nourished communities the mortality is generally as low as 4-8 per cent., but under famine conditions and in debilitated persons it may rise to 40 or even 50 per cent. The bilious-typhus type, taken by itself, gives a mortality as high as 70 or even 80 per cent. In African tick fever the mortality among Europeans is rather high, and also among natives from non-endemic areas. Signs of bad prognostic import are hyperpyrexia, cardiac weakness, low nervous symptoms, severe hæmorrhages, and severe abdominal symptoms.

**Prophylaxis** must be directed to stamping out the insect carrier. Lice are readily killed by boiling the clothes or even by simple washing in antiseptics followed by sun-drying but best of all by one of the methods of steam sterilization. Travellers may avoid African tick fever by keeping clear of native huts and old camping grounds, which are apt to be infested with ticks.

**Treatment.**—Salvarsan and neosalvarsan have a specific action on the spirochaete; from 0.3 to 0.5 gm. of the latter (as being less toxic) should be injected intravenously. One dose will generally bring about an artificial crisis with relief of all symptoms, and relapses are generally prevented. Otherwise, a second dose may be required.

Conseil has recommended two other arsenical compounds, galyl and ludylyl, in doses of 0.3-0.5 gm., as being less toxic and even more efficacious than neosalvarsan. Extensive experience in different parts of the world during the War has fully borne out the great value of salvarsan and its derivatives as a specific in this group of diseases. F. PERCIVAL MACKIE.

**REMEDIAL EXERCISES.**—Like all other forms of physico-therapeutics, Remedial Exercises have received a vastly increased recognition through the experience gained in the War.

The idea of using movements as a curative agency may fairly be claimed to have originated



## REMEDIAL EXERCISES

with the Swede, P. H. Ling (1776-1839), and it has been spread by his successors and followers practically all over the world, to be adopted and modified with more or less readiness and success in various countries. Thus, in England it has been followed up with an increasing degree of enthusiasm and thoroughness for quite a generation, yet the value of this form of treatment would not have been brought into prominence so rapidly except for the events of the last few years.

It was from the exercises which Ling invented, or, more correctly speaking systematized, for healthy individuals, that the movements used for remedial purposes gradually developed. At first, no doubt, these were merely *active* movements, i.e. such as are carried out by the patient's own voluntary effort, with or without resistance; but later they included *passive* ones performed by an operator with or on the patient's body, for Ling included under passive movements all the various massage manipulations used by him. A thorough acquaintance with and personal experience of the ordinary Swedish gymnastics, such as are now taught in most schools, and have recently been introduced into the Navy and Army, are the best foundation for a full understanding and intelligent administration of remedial exercises. It will then be appreciated that the object of the exercises is not primarily to produce great muscular power, but to further the proportionate and harmonious development of the body, and thus to produce the form of strength which results in increased health and feeling of well-being.

Turning now to the practical application of these principles in the correction or prevention of pathological conditions, their great value in dealing with **deformities** comes readily to mind. One of the most important fields of usefulness of remedial exercises in this connexion is in the treatment of *spinal curvatures*. In addition to various other measures, such as rest supports or corrective jackets and stimulation of muscles by massage or electricity, which may be employed in such cases, it is almost certain that at some stage the aid of exercises will have to be invoked.

Their object is to mobilize the spine, stretch contracted muscles and ligaments on the concave side of the curvatures, and strengthen the relaxed and weakened muscles on the convexities. To this end, such positions should be chosen as enable the weight of the body to help in correcting the deformities and give the

greatest advantage to the action of the muscles in the particular section of the spinal column. In spinal curvature, special attention ought always to be given to the development of the chest, as this generally shares more or less in the deformity. Moreover, the improved action of the lungs will very often help not only to improve the shape of the thorax but also to influence the deformities of the spinal column. The same guiding principles apply in the treatment of various other deformities, whether congenital or acquired.

*Torticollis*, for instance, is a condition that often coexists with spinal curvature, either as effect or as cause, and frequently comes under treatment by remedial exercises with or without previous operation. The plan, then, should be, first to correct, or rather to over-correct, the position of the head by manipulations, then to make the patient maintain the corrected position to the best of his ability by his own muscular effort, later to bring his head into that position himself, and finally to do so against resistance. An important help is to let him perform in front of a glass so that the sight may guide him to an appreciation of what the position of the head should be, as his own notion of a correct attitude is likely to have become altogether perverted.

A great variety of deformities of the extremities lend themselves to similar treatment, especially static deformities of the lower extremities. Thus, in *congenital dislocation of the hip-joint* there is sure to be a stage in the treatment, i.e. after the child has been taken out of its plaster-of-paris, when judicious exercises form an indispensable part of the régime for restoring the function of the limb as far as possible. In this instance it is, however, important not to be ambitious to restore the greatest possible degree of mobility, as flexion and adduction of the hip-joint increase the risk of a recurrence of the dislocation. It is the extensors and abductors that should be brought into use and strengthened, as their action tends to help in maintaining the head of the femur in the acetabulum and to enable the patient to stand and walk upright. Thus the necessary opportunity is given for that functional adaptation of the various parts of the joint upon which the success of the whole treatment of such a case ultimately depends.

*Coxa vara* is another example of deformity affecting the hip-joint in which remedial exercises, often preceded by osteotomy to correct the position of the femur, are of the

## REMEDIAL EXERCISES

utmost importance. Their object here is to restore the greatest possible degree of movement in the joint; and although unfortunately this often remains permanently extremely limited, a much more useful limb can be obtained by training the patient to take full advantage of the mobility that can be regained in the hip and to use his knee and ankle to make up for the deficiency.

Certain deformities in connexion with the *knee-joint* respond more readily to exercises as they are more directly due to muscular weakness and are enhanced by the body-weight being transmitted in an incorrect manner to a loose and insufficiently supported joint. The best example of such a deformity is *genu valgum* of the adolescent type. Much can here be accomplished by the practice of exercises that strengthen the extensor quadriceps, especially the internal vastus, such as bending and stretching the knee, the latter motion with resistance, and preferably with pressure administered by the operator on the inner side of the knee and outer side of the ankle, so as to make the joint work as far as possible in the correct line. Attempt at voluntary abduction of the thighs, with the knees kept straight and the ankles held or tied together, is another useful exercise in such a case; and the patient ought to be made to stand and walk habitually with the feet turned more or less parallel and kept as near together as possible. Further, riding a pony is obviously a more suitable occupation for such a youth than riding a bicycle.

The various types of *talipes* constitute another large group of deformities where remedial exercises form an important part of the treatment, often certainly as a mere adjunct to surgical and instrumental measures, but in milder cases proving in themselves a sufficient corrective. A slight degree of *pes valgus*, and still more the earlier stage of this condition, when there is merely eversion of the feet, can thus be very successfully dealt with by passive and active movements that stretch the peronei and at the same time strengthen and contract the "supinators" of the foot, the *tibiales anticus* and *posticus*. Circumduction of the foot, or as it is technically termed "foot-rolling," forms one useful exercise for this purpose, but before this can be carried out actively by the patient it ought to be done passively by an operator, who takes care that the movement is carried to its full extent in the inward direction, so that the peronei become fully stretched. In addition, the patient

should practise inversion of the foot against the operator's resistance, and the latter then brings the foot back again while the patient resists, but with this movement carried to the mid-line only. The description of this simple performance is here given in detail, in order to illustrate two terms and principles that are constantly applied in remedial exercises. The first is the *concentric and eccentric* muscle action, which is obtained when the two *tibialis* muscles first contract against the operator's resistance and then gradually give way to his pulling the foot outwards. The second is, that when the operator only carries the foot as far as the mid-line, but the patient brings it inwards to the fullest possible extent, the same muscles are made to work in the *inner half of their path of contraction*. This tends permanently to shorten them, whereas action in the *outer half* of a muscle's path of contraction helps to make it longer; a circumstance of which advantage is often taken in arranging exercises.

Much of what has now been said about the treatment of deformities applies also to conditions resulting from various kinds of *injuries*. The principles are the same as those already laid down: contracted structures should be pulled upon and stretched; weakened muscles exercised and strengthened, and the functional utility of the injured part restored.

In any scheme for accomplishing this purpose, the natural action of muscles in response to the voluntary nervous impulse of the patient must always be the chief or, rather, the ultimate factor. Only thus can the most rapid and complete recovery of which circumstances permit be achieved. This applies not only to the accidents common in civilian life but also to an overwhelming number of the injuries met with during the War; it is in this connexion that remedial exercises as well as other physical therapeutics have achieved so much during the last few years.

It is, however, not merely in dealing with pathological conditions of an external nature that remedial exercises are valuable. Numerous *diseases of internal organs* can be very favourably influenced by them. Thus the improved shape and mobility of the thorax as a result of exercises must influence powerfully the organs contained within the thoracic cavity. Various affections of the lungs and pleura are, as a matter of fact, greatly benefited by suitable exercises, though, except in some chronic conditions, their scope of usefulness may be only as after-treatment, or as a

## REMEDIAL EXERCISES

preventive measure. Among such chronic conditions may be mentioned *emphysema* and *chronic bronchitis*. In the former affection it is of course to expiration that particular attention has to be paid and the exercises can quite well be adapted accordingly. Simple *pleurisy* and *empyema* serve as examples of diseases in which exercises come in as valuable adjuncts in the after-treatment, the object then being to restore the normal mobility of the thorax by means of trunk exercises and deep breathing so as to prevent or break down pleural adhesions and to make the lung expand again. After empyema, with resection of some portion of the chest-wall, exercises are of great importance in preventing deformity of the thorax and spine as well as in restoring the air-entry in the collapsed lung. The proper development of a *phthinoid chest* constitutes an instance of the great value of appropriate breathing exercises as a prophylactic measure, whereas such exercises have to be practised with the utmost caution in any case of established pulmonary tuberculosis, for fear of bringing on a hæmorrhage or of causing a flare-up of a safely encapsuled focus in the lung.

Various derangements of the *cardio-vascular system* benefit also in a marked manner by remedial exercises. The chief ways in which the heart can thus be influenced and helped in its work are: (1) By aiding the peripheral circulation in veins and lymph-vessels; for this purpose, passive and also active movements are used with advantage in combination with various massage manipulations. (2) By deep-breathing exercises which help the circulation through the lungs, the aeration of the blood and the diastole of the heart. (3) By a gradual training of the heart-muscle itself by means of carefully graduated exercises for the extremities and trunk which give it a moderate but progressive amount of work.

This latter idea forms the key-note of Oertel's so-called "*terrain-cure*," which consists in walking according to a regular prescribed scale certain stated distances and up selected slopes. In the famous *Nauheim treatment* we find incorporated the first as well as the third of the above-mentioned principles. The second is, however, entirely neglected, in that all the resisted exercises advocated are performed at a very slow rate, each individual movement occupying about a minute and not being repeated. In the Swedish School, on the other hand, the various movements are repeated a number of times (3-10) according to the patient's

condition, and, whenever possible, are adapted to the rate of breathing. This has always been considered an essential point, contributing greatly to the success with which heart affections have been treated by such methods in Sweden.

The organs of the *abdominal cavity* are naturally greatly influenced by exercises, especially by those of the trunk which bring into action the muscles of the abdominal wall, which are often much neglected. The circulation through the liver and the portal system generally is much improved by the alternating pressure thus brought to bear upon it; and obviously this must be of considerable value in conditions causing or resulting from a congested state of the liver. Further, the intrinsic muscles of the stomach and intestines gain in nutrition and strength by the increased circulation through them, and so the peristaltic action is improved with great benefit to the function of the digestive organs. *Chronic constipation* is the digestive disorder which most obviously comes to mind as likely to respond to treatment by exercises, but it is by no means the only one. *Indigestion*, *chronic catarrhal gastritis*, *gastric dilatation*, and *visceroptosis* are among the affections that may with advantage be treated in this way.

Even the organs of the *pelvic cavity* are not beyond the influence of remedial exercises. The pressure from powerful contractions of the abdominal muscles is naturally transmitted to the pelvic viscera as well as to those of the abdomen; and so is the effect of the deeper descent of the diaphragm which is produced by breathing exercises. Further, the circulation through the pelvic viscera can be influenced considerably by the proper choice of exercises. Thus, contractions of the large flexors of the hip-joint (ilio-psoas) and of the abdominal muscles will send an increased supply of blood to the pelvis, whereas action of the hip extensors, especially the glutei muscles, and of the erector spinæ in the lumbar region will tend to draw the blood from the pelvis. This effect of the appropriate exercises is often technically referred to respectively as "repletive" and "depletive."

The best known application of exercises in connexion with *nervous diseases* is probably the method of treating inco-ordination in *locomotor ataxia*, which is known as Frenkel's exercises. Frenkel classifies them into those performed in (1) the lying position, (2) sitting, (3) standing, (4) walking, and (5) exercises for

## REMEDIAL EXERCISES

the upper extremities. They consist in placing the limbs in certain positions or moving them along certain directions either in a definite fixed order or according to command, and this partly with the aid of sight and partly with the eyes shut. Thus, by very simple means, a series of movements is provided, carefully graduated so as to demand more and more precision and co-ordination in their execution, and persevering practice will improve, sometimes in a remarkable degree, the patient's power and control over his movements.

It is not only in tabes dorsalis that such exercises are of value. *Disseminated sclerosis* with its uncertainty of movement is another disease in the treatment of which they may be distinctly beneficial. With suitable modifications, exercises are used with advantage in a great variety of affections in the central nervous system. Another example, though one of a different type, is *hemiplegia*. The ultimate object of the exercises in this affection is to restore, as far as possible, the control of the higher centres and to enable voluntary impulses again to reach and control the muscles. To facilitate such a recovery, attention must first be given to the prevention of contractures and to the maintenance of the muscles in the best possible condition, so as to keep them in a state responsive to voluntary stimuli. To this end passive movements (in addition to massage) are of the greatest importance. Great caution has, however, to be observed, in all cases of which spasticity is a leading feature, to avoid producing spasm by any sudden and abrupt manipulations and movements. On the other hand, it is surprising how apparently rigid contractions, as in *spastic paraplegia* from birth injury, will relax to a gentle but sustained pull or pressure.

Among lesions of the lower motor neurones none is probably more frequently treated by remedial exercises than the varied conditions resulting from *poliomyelitis*. Whatever other measures may be employed in dealing with this affection, according to the circumstances of the case or the judgment of individual authorities (such as complete rest or, on the contrary, stimulation by massage and elasticity, the use of instruments, and operative procedures such as tenotomies or tendon- and nerve-transplantations), it is certain that sooner or later exercises will have to be resorted to. Only by their aid can the healthy portions of the damaged muscles be preserved, their remaining power cultivated, and other muscles trained to

## RESPIRATION, ARTIFICIAL

perform the deficient movements, thus preventing or limiting disabilities and deformities. It is a kind of treatment that requires great judgment, perseverance, and patience, but, if it is employed at the right time and in the right manner, much of the operative and instrumental treatment necessary later on in neglected cases might be avoided.

One of the most important of the uses of exercises during the War was in the treatment of *peripheral nerve affections*. In an immense number of such cases the final recovery, more or less complete, and the success of the surgeon's work in the way of sutures, anastomoses, and nerve-transplantations, were only made possible by the aid of appropriate exercises.

RICHARD TIMBERG.

**REMITTENT FEVER** (*see* RELAPSING FEVER).

**RENAL CALCULUS** (*see* URINARY CALCULI).

**RENAL COLIC** (*see* COLIC; URINARY CALCULI).

**RENAL FUNCTION** (*see* NEPHRITIS).

**RENAL SYPHILIS** (*see* NEPHRITIS).

**RENAL TUBERCULOSIS** (*see* KIDNEY, TUBERCULOSIS OF).

**RENAL TUMOURS** (*see* KIDNEY, TUMOURS OF).

**RESPIRATION, ARTIFICIAL.** 1. **Sylvester's method.** - The patient is placed on his back, his head being allowed to hang downwards over the end of the bed or table or, if he is on the ground, over a roll of clothes placed beneath his shoulders. It is advisable, if the means are at hand, to pull out and make traction on the tongue by tongue forceps, or the tongue may be grasped and pulled forward with a pocket-handkerchief. The operator should stand behind the patient, and, grasping the arms at the elbows, should press them firmly and steadily against the sides of the chest for about two seconds. This produces expiration. If it is ineffectual it may be reinforced by pressure below the costal margins, directed towards the diaphragm. Afterwards the arms should, slowly and deliberately, be brought towards the operator, keeping the elbows close to the ground, so that they come to lie in the long axis of the patient's body, one on each side of the head. They should then be pulled on steadily for two seconds.

## RESPIRATION, ARTIFICIAL

By this means the chest is expanded and inspiration effected. These two movements should be repeated rhythmically and alternately, about fifteen times a minute. When normal respiration returns it should be supplemented by the artificial movements until satisfactorily re-established.

**2. Schäfer's method.**—The patient is placed face downwards on the floor, the head being turned to one side. Kneeling astride or to one

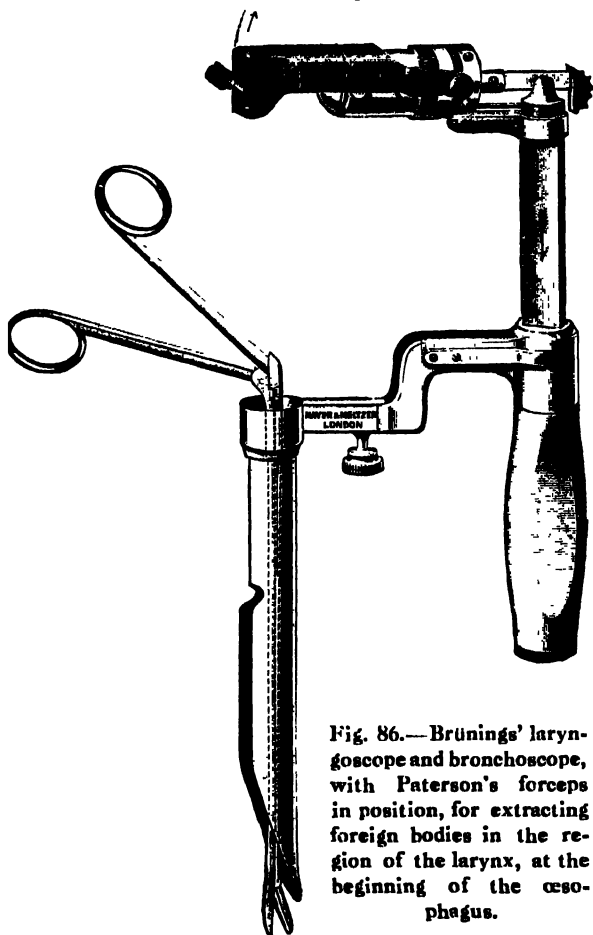


Fig. 86.—Brünings' laryngoscope and bronchoscope, with Paterson's forceps in position, for extracting foreign bodies in the region of the larynx, at the beginning of the oesophagus.

side of the loins, the operator places his hands on the patient's back, the thumbs being parallel to the spine, and about 3 in. apart, and the fingers spread out over the lower ribs on each side. To produce expiration the operator leans forward, throwing the weight of his body on to his hands. He then leans back, taking the weight off his hands, but keeping them in position. The elasticity of the patient's ribs expands the chest and induces inspiration. The

## RESPIRATORY PASSAGES

movements are repeated about twelve times a minute.

**3. Chest compression.**—The patient being on his back, the operator's hands are spread out over the lower ribs on both sides. Pressure is exerted at regular intervals approximating to the normal respiratory rate. This simple procedure may be all that is necessary if used directly after respiration has ceased, especially in the case of children.

**4. Laborde's method.**—This is performed by rhythmically pulling on the tongue by means of tongue forceps.

**5. Schultze's method,** which is sometimes employed in asphyxia neonatorum, is far from safe, and is not recommended.

FREDERICK LANGMEAD.

## RESPIRATORY PASSAGES, ENDOSCOPIC EXAMINATION OF.

—This procedure is undertaken for—

1. Examination.
2. Removal of foreign bodies.
3. Treatment of local ulcerations, stenosis, etc.

The instrument now invariably used is Brünings' laryngoscope and tracheo-bronchoscope (Fig. 86). The patient should, if practicable, be prepared, the mouth being rendered as clean as possible and no food being taken for eight hours before the passage of the instrument, whether this be done under local or general anaesthesia. When the case is one of impaction of a foreign body, an immediate X-ray examination should be made, promptly followed by endoscopy—which should only be undertaken by one who is an expert in the method.

**Anæsthesia.**—For purposes of examination in the adult, *local* anæsthesia under cocaine answers admirably. The soft palate and pharynx are sprayed with a 5-per-cent. solution of cocaine; after a few minutes a laryngeal mirror is used, and under direct observation a 15-per-cent. solution of cocaine is applied on a bent swab-holder to the base of the tongue, both surfaces of the epiglottis, and the surface of the larynx. This usually has to be repeated twice or thrice.

For examination in children, and in all cases of treatment and removal of foreign bodies, a *general* anæsthetic is required. Chloroform is preferable to ether, as the latter causes excessive salivation. In any case, atropine sulphate

## RESPIRATORY PASSAGES

$\frac{1}{10}$  to  $\frac{1}{100}$  gr. should be given hypodermically an hour before the undertaking, and when possible a mixture of bromide and belladonna administered thrice daily for three to four days; this tends to lessen the irritability of the mucous membrane and diminish the secretions.

**Method of passing the instrument.** 1. **Under local anæsthesia in the adult.**—The patient should be seated on a low stool with an assistant on a higher stool directly behind. This assistant steadies the head, which is thrown right back, and he also controls a Doyen's gag by which the mouth is kept open. The surgeon stands to the right of the patient and slightly in front. The instrument is taken in his right hand, and, the light having been switched on, is carefully passed down over the tongue until the epiglottis is seen; it is now passed over the epiglottis for a distance of half an inch. Then by a gradual down-turn of the wrist the epiglottis and base of the tongue are steadily pushed forward. This will bring the larynx directly under view. Care must be taken not to use the front incisor teeth as a fulcrum in this movement.

Many surgeons use local anæsthesia and the above position for examination of the trachea and bronchi in adults. For this purpose a tracheo-bronchoscope is attached to Brünings' handle, and the technique for the first part of the procedure is exactly as above. The larynx being brought into view, the operator waits till the patient takes an inspiration, when, on the cords abducting, the tube is slipped in between them. This invariably induces considerable coughing, but a swab soaked in a 10-per-cent. solution of cocaine is at once passed down the tube and into the trachea; in a few seconds this allays all irritability, and the examination can then be completed.

2. **Under general anæsthesia.**—The patient is placed on a high operating table in the dorsal decubitus, and when he is completely under the influence of the anæsthetic the head is lowered over the upper edge of the table and supported by an assistant sitting to the left. The procedure is the same as above.

When the trachea and bronchi are examined by the above method the name *upper tracheo-bronchoscopy* is applied to the proceeding. This is in contradistinction to *lower tracheo-bronchoscopy*, which is undertaken through the opening in the trachea subsequent to a tracheotomy.

The following points are important: Always

## RETAINED CONCEPTION PRODUCTS

have tracheotomy instruments at hand. In doing a tracheoscopy or bronchoscopy, always use the widest tube possible; and it is well to remember that in the adult the measurements of the glottic aperture, the trachea, and the bronchi are—

Glottic aperture . . .	12 × 22 × 22 mm.
Diameter of trachea . . .	14 × 20 mm.
Length " " . . .	12 cm.
" , right bronchus . . .	2·5 cm.
" " left " . . .	5 cm.
" from incisors to trachea	15 cm.

In women these measurements are slightly less, and in children much smaller, depending on the age.

J. GAY FRENCH.

**RETAINED PRODUCTS OF CONCEPTION.**—A result of incomplete abortion in the early months of pregnancy, before the full formation of the placenta. The products retained may include everything but the foetus, or only small masses of decidua or forming placenta, all else having escaped from the uterus.

**Etiology.**—The frequency of retention is explained by the thickness and friability of the decidua, which resist separation from the uterine wall when contractions occur, whereas the ovum is soft and compressible and comparatively readily detached.

**Symptomatology.**—In uninfected cases the chief symptom is hæmorrhage. This varies extensively in amount and quality. Bleeding may be so free as to endanger the patient's life, or may appear only as an irregular brown discharge containing fragments of solid tissue. In infected cases, which are usually due to interference from without, there is rise of temperature and of pulse, tenderness of the uterus, or more general tenderness in the lower abdomen, and purulent offensive discharge mixed with the blood, which escapes. Just as in infection after a full-term labour, all the more remote signs of sapræmia and septicæmia may show themselves.

**Diagnosis.**—In all cases the bladder and lower bowel should be empty when examination is made. Diagnosis in many cases, especially recent ones, is simple. The patient gives an account of one or two periods missed, an attack of pain and hæmorrhage, and can frequently show what has come away. On examination the uterus is found softened and enlarged, the cervix soft and patulous, blood and clots are present in the vagina, and sometimes a mass can be felt through the open cervix. The fornices are clear.

## RETAINED PRODUCTS OF CONCEPTION

In other cases, in view of the possible variation in size and consistency of the uterus, according to the mass of tissue left in it, and since hæmorrhage may be caused by retained products of conception long after abortion has taken place, diagnosis is difficult. In some instances, especially when examination of the posterior and lateral vaginal fornices reveals the presence of a mass, differential diagnosis may be of first importance. The history may be so indefinite as to be of little or no value. Cases in which diagnosis is difficult may be separated into the following groups:

1. *Those in which there is bleeding from the uterus and no mass in the posterior or lateral fornices.*

(a) Pregnancy may be progressing in spite of loss. The diagnosis is especially difficult when the uterus is retroflexed. It is made by considering the history in conjunction with the size of the uterus and, in case of doubt, immobilizing the patient and watching the progress of the case.

(b) Carcneous mole. In contrast to the above, the uterus tends to shrink. Diagnosis is confirmed only by exploration of the uterus.

(c) Retained portions of a hydatidiform mole or early chorion-epithelioma. Vesicles may be found in the vagina or felt through the os on examination. Otherwise the diagnosis can be made only by exploration of the uterus.

(d) Fibroid or mucous polypus of the uterus. The history usually helps considerably. The loss consists of no other solid matter than clot. A uterus containing a fibroid polypus may contract and relax like a pregnant uterus.

(e) Generalized fibroid, sarcoma, or carcinoma of the uterine body. The history again is helpful. Carcinoma of the body usually occurs after the menopause, and most frequently in those who have not borne children.

2. *Cases where there is bleeding from the uterus and a mass in one or both fornices.*—The mass may be associated with pregnancy or be independent of it. The bleeding from the uterus may result from the disease which produces the mass.

The chief disorders that come under this heading are:

(a) Extra-uterine pregnancy.

(b) Extra-uterine with intra-uterine pregnancy.

(c) Salpingo-oöphoritis with or without early abortion.

(d) Retained products of conception with ovarian or other cystic tumours.

In all cases of doubt, intra-uterine manipulation should be avoided, and the patient transferred to a suitable nursing home or hospital where abdominal section can be undertaken if required.

**Prognosis.**—The prognosis is good, except in those cases which have become infected. Vigorous treatment may have to be undertaken to stop hæmorrhage, but the response to operative measures is very satisfactory. The outlook in infected cases is serious.

**Treatment.**—With the doubtful exception of certain septic cases, the essential part of treatment lies in the removal of the retained fragments. In recent cases where the cervix is patulous, this may be done manually and without an anæsthetic, though even here anæsthesia is desirable. Most cases, however, definitely require an anæsthetic, dilatation of the cervix, and removal of the retained products instrumentally. Unless the parts are so dilatable that one finger, preferably two, can readily be introduced into the uterus, instrumental removal of the fragments is more satisfactory than their manual removal. The patient is anæsthetized, placed in the lithotomy position, and the external parts prepared for aseptic operation by washing, shaving, and disinfection; tincture of iodine is frequently used for this purpose. The vagina is douched with anti-septic lotion (lysol, 30 min. to the pint, is useful) or swabbed, and then treated with tincture of iodine. A final examination is made, the size and position of the uterus being especially noted. Rubber gloves are worn. A speculum is then introduced into the vagina (Auvard's weighted speculum is of great value when assistance is limited), the anterior lip of the cervix is seized with volsella forceps and the cervix dilated with graduated dilators. (If the masses inside the uterus are large the parts are usually readily dilatable; if small, there is no need for great dilatation—it is then only necessary to pass small ovum forceps into the cavity.) Ovum forceps should then be introduced into the uterine cavity, and its vault, sides, cornua, anterior and posterior surfaces methodically examined. The forceps are opened at these various sites and then shut and withdrawn from the uterus with a twisting movement. No strong tension should be applied, as the soft uterine wall or a dependent polypus may have been seized by mistake. The process should be continued until the whole of the interior surface of the uterus has been explored. For this the instrument has

## RETAINED PRODUCTS

usually to be introduced many times. A sharp flushing curette is then used—less harm is liable to be done with a sharp curette used carefully than a blunt one used, as it must be, more forcibly—and the whole surface very lightly gone over, the cavity being at the same time flushed out with weak lysol solution at 120° F., to stimulate contraction and prevent further bleeding. With this method any fragment still retained can be felt from the irregularity it produces, and recourse may again have to be had to the ovum forceps. Before the douche is withdrawn from the tract the vagina also is douched. Pituitrin (1 c.c.) may be given intramuscularly if contraction is not satisfactory. Packing of the uterus is rarely necessary. If the patient had lost seriously before operation, subcutaneous saline (2-3 pints) should be injected under the breasts throughout the operation. Iron and extra fluids should be given during convalescence.

The decision as to what treatment to adopt in septic cases is a matter requiring considerable judgment. Stirring up of the septic contents of the uterus is to be avoided as much as possible; curettage of its walls is strictly contraindicated. Yet if it is obvious, from the tenderness and increased size of the uterus, or from purulent and offensive discharge, that it contains masses of septic material, there is a call for manipulative treatment.

Under anæsthesia, first a specimen of the uterine contents should be taken for bacteriological examination and the preparation, if thought desirable, of an autogenous vaccine. Then the cavity should be very gently explored with ovum forceps, and any masses removed. A copious mild antiseptic douche at low pressure should now be given, and if drainage is not very free through the cervix, a wide drainage-tube, attached to the cervix by a catgut stitch, should be introduced to keep the cervix patent. The patient should remain in bed in the Fowler position, ergot combined with quinine and strychnine given by mouth, and general strengthening treatment adopted. The bowels should be kept freely open daily, rather by the use of enemata than by any drastic purge.

When the uterus does not show definite signs of containing septic matter, or the septic process has spread into the broad ligament or the peritoneal cavity, expectant treatment should be adopted.

FRANCES M. HUXLEY.

## RETENTION OF URINE

**RETENTION OF URINE.**—Urine may be retained in the bladder either as the result of obstruction to the urethra or failure of the detrusor mechanism.

The obstructive causes are largely surgical, and include stricture, calculus, prostatic enlargements, whether inflammatory or due to benign or malignant growths, new growths of the bladder, retroflexion of the gravid uterus, congestion of the urethra as in gonorrhœa or after injuries, and phimosis or a pinhole meatus in children. In an important group the sphincter itself provides the obstruction by spasm or by failure to relax. This may occur reflexly after catheterization or the passage of other instruments, after operations in the pelvis or on the rectum, and after parturition. Local damage to the urethra is difficult to exclude in many of these instances. The retention of hysteria is probably due to sphincter spasm, as also is the inability of nervous persons to micturate in the presence of others.

Normal micturition is a voluntary act, contraction of the detrusor muscle and relaxation of the sphincters being excited by cortical impulses. When these are interfered with by bilateral cerebral lesions or transverse lesions of the cord, retention follows. That which accompanies shock, and coma resulting from intracranial pressure or profound toxæmia, is similarly explained. Partial or complete retention is a symptom of many nervous diseases. It occurs particularly in local and systemic diseases of the spinal cord; it is common in the early stage of poliomyelitis and in tabes dorsalis, of which it seriously affects the prognosis should catheterization become necessary. In the latter disease loss of sensory impulse from the bladder is its probable explanation. (The relations between the nervous system and the mechanism of micturition are dealt with more fully under INCONTINENCE OF URINE.)

Failure of the detrusor action of the bladder occurs in old people and in prostrating disease such as typhoid fever. Its most common antecedent, however, is overstretching of the bladder-wall when the outflow of urine is hampered by obstruction.

The recognition of retention is not difficult from the history and the presence of acute discomfort or pain in the lower abdomen and of a distended bladder, but it may be overlooked when nervous lesions produce anæsthesia of the bladder. Whether it is due to organic obstruction is decided by the passing



## RETINA, AFFECTIONS OF

of a catheter in those cases in which the nature of the disorder is not sufficiently obvious by local or rectal examination. It must be remembered that a suprapubic examination should be made in all cases of incontinence of urine, lest the distended bladder be overlooked and retention with overflow be regarded as true incontinence.

The **treatment** is that of the cause. That due to spasm of the sphincter will generally yield to fomentations above the pubis or to a warm bath, whilst in hysterical cases a strong hydragogue purge is usually efficacious as a preliminary to the treatment of the hysteria. The treatment of the retention due to organic obstruction is considered under its various causes. In retention due to organic nervous disease or to atony of the bladder catheterization should be avoided as long as possible, and, when it is necessary, scrupulous asepsis must be practised (*see* CATHETERIZATION). It is frequently possible to obtain natural evacuation of the bladder by placing the patient in a more favourable position, or by a fomentation or pressure over the pubis. In these cases it is most important not to allow the bladder to become over-distended, as the more the muscles of its walls are stretched the more they lose their efficiency.

FREDERICK LANGMEAD.

**RETINA, AFFECTIONS OF.**—Under this title are included—

1. RETINITIS.
2. HÆMORRHAGES OF THE RETINA.
3. EMBOLISM OF THE CENTRAL ARTERY OF THE RETINA.
4. RETINITIS PIGMENTOSA.
5. DETACHMENT OF THE RETINA.
6. GLIOMA OF THE RETINA.

### 1. RETINITIS

This term is loosely applied not only to inflammatory but also to degenerative and other pathological changes in the retina. The degenerative changes are often secondary to inflammation or other abnormality of the choroid, for owing to the dependency of the outer layers of the retina on the choroid for their nutrition, choroiditis invariably causes retinal changes. On the other hand, there are several forms of primary retinitis. The latter group is best termed retinitis, the former choroido-retinitis (*see* Choroiditis, under UVEAL TRACT, AFFECTIONS OF).

Retinitis is usually classified according to its

etiology. **Syphilitic retinitis** is one of the commonest types; it may be either a true primary retinitis or a choroido-retinitis, and the latter is the more frequent. Primary syphilitic retinitis causes obscuration of vision, largely due to dust-like opacities in the vitreous, especially the posterior part. The retina looks dull and opaque with the ophthalmoscope, and the disc abnormally red. There may be small yellowish or white spots in the macular region, and others, often surrounded by pigment, in the periphery. The vessels are usually affected with syphilitic endovascular changes, and may be bounded by white lines. Hemorrhages are uncommon. Extensive patches of pigmentary degeneration, in some cases almost indistinguishable from retinitis pigmentosa, may be formed. In these cases metamorphopsia, scotomata, irregular contraction of the field of vision, and night-blindness may be added to the defective central vision. Sometimes the macula alone is attacked; there is a greyish-yellow deposit in this situation, accompanied by metamorphopsia, and followed by pigmentation and a central scotoma. There is a definite tendency to the formation of new blood-vessels in syphilitic retinitis; these often constitute small convoluted bunches, which may project into the vitreous, and are held together by films of connective tissue (retinitis proliferans). The disease usually occurs one or two years after infection. The second eye is generally affected after a variable interval.

In congenital syphilis, retinitis usually takes the form of stippled pigmentation, especially at the periphery, often associated with discrete white spots, like a mixture of pepper and salt.

**Diagnosis and treatment.**—The history and the Wassermann test usually suffice to establish the diagnosis. Rest, dark glasses, and anti-syphilitic treatment are indicated.

**Albuminuric retinitis** (PLATE 23, Fig. 4, Vol. II, facing p. 412) occurs especially in cases of chronic interstitial nephritis and in the nephritis of pregnancy; less frequently in other forms of nephritis, but very rarely in acute nephritis. The typical ophthalmoscopic picture is almost pathognomonic, though it may occur in some cases of intracranial tumour. There is optic neuritis, with comparatively slight swelling of the disc, associated with white exudates in the retina. The latter form a fan- or star-shaped figure at the macula, composed of radiating lines of brilliant white spots. The whole area of the retina around the macula

## RETINA, AFFECTIONS OF

and disc is hazy and is studded with soft-edged cloudy-white exudates and small hæmorrhages. The disc is often surrounded with large white patches or a continuous "snow-bank." The blood-vessels nearly always show signs of vascular disease—silver-wire arteries, irregularity of contour of the veins, constriction of the veins where arteries pass over them, white lines due to thickened walls, etc. These may be entirely absent in the pregnancy cases. In the latter a flat detachment of the retina may be produced by masses of subretinal exudation. When these are absorbed the retina returns to its normal position.

Retinitis associated with albuminuria by no means always shows the typical picture. If the star at the macula is absent the ophthalmoscopic appearances may be indistinguishable from those of diabetic retinitis. In other cases there is a simple neuro-retinitis, with slight swelling of the disc, blurred edges, and widespread œdema of the retina and hæmorrhages. There are usually scattered spots of exudate, often near the vessels.

The typical picture is not directly due to the vascular disease but to toxins, and the prognosis for life is very grave, the patient rarely living more than two years. This does not apply to the pregnancy cases. In these the prognosis for vision depends on the duration of the retinitis. On this account, and owing to the danger of eclampsia, the artificial induction of abortion is generally indicated. Vision is usually permanently impaired, due to partial optic atrophy accompanied by white or pigmented spots in the macular region. The albuminuric retinitis of pregnancy does not always occur at the first pregnancy, and though it generally recurs at subsequent pregnancies, this is not an invariable rule.

The albuminuric retinitis of chronic renal disease is commonest between 50 and 60 years of age. It is nearly always bilateral, and is associated with severe headaches and high blood-pressure. It rarely causes complete blindness. The treatment is that of the underlying cause.

**Diabetic retinitis** (PLATE 23, Fig. 5, Vol. II, facing p. 412) is rather rare. It is characterized by irregularly scattered bright white spots in and around the macular region. Small round hæmorrhages are distributed over the fundus; they are more deeply seated than the minute flame-shaped hæmorrhages common in renal retinitis. Diabetic retinitis occurs chiefly in the late stages and in elderly people.

It is often accompanied by albuminuria, and the ophthalmoscopic appearances may be correspondingly complicated. Apart from renal disease, the vessels are usually fairly healthy and the prognosis for life is much better. From associated lipæmia the retinal vessels may appear to be filled with fluid resembling milk; the arteries are pale red, and the veins violet.

**Leukæmic retinitis** has a characteristic ophthalmoscopic picture. The fundus is pale and orange-coloured. The arteries are small and pale red; the veins bright red, dilated and tortuous, often with white lines along them. White spots with a reddish border, composed of leucocytes surrounded by red corpuscles, are occasionally found scattered about the fundus; these occasionally occur in pernicious anæmia. Leukæmic retinitis is rare; more commonly the retina is normal, or only shows ordinary retinal hæmorrhages.

**Purulent retinitis** is a septic condition due to a perforating wound of the eye or, more rarely, to metastasis. In the early stages there is much œdema of the retina with hæmorrhages. The retina rapidly becomes infiltrated with pus cells, and looks swollen and red. In the later stages there is a yellow reflex from the pupil, especially when the pus invades the vitreous and the condition passes into panophthalmitis. This is rarer in metastatic endophthalmitis, in which, owing to the attenuated virulence of the organisms in the blood and tissues, subsidence of the inflammation may occur and even useful sight be restored.

**Retinitis from exposure to bright light** occurs typically in "eclipse blindness," when the sun is viewed with imperfectly protected eyes. It may also be caused by the arc light or the flash of a short circuit. The normal after-image persists, passing into a positive scotoma, which may be permanent. In the early stages there may be metamorphopsia, due to disturbance of the cones at the fovea. The macula may look normal immediately after the exposure, or may show a pale spot at the fovea, surrounded by a reddish-brown ring. Gradually spots of pigment appear in this region. The prognosis varies with the intensity and duration of the stimulation and the individual patient. All stages, from an unduly persistent after-image to a permanent positive central scotoma, are met with.

For *hæmorrhagic retinitis*, *retinitis proliferans*, *exudative retinitis*, and *retinitis circinata*, see the next section.

## RETINA, AFFECTIONS OF

### 2. HÆMORRHAGES OF THE RETINA

These hæmorrhages are due to many causes. They may occur with normal retinal vessels, as in whooping-cough, pressure during birth in new-born infants, severe compression of the thorax in crowds, etc. They may also be due to direct injury, such as blows upon the eye, perforating wounds, and gunshot wounds in the neighbourhood of the eye. They may follow an intra-ocular operation; in these cases, owing to the sudden diminution of the intra-ocular pressure produced by the operation wound, the ocular vessels dilate and may be insufficiently strong to resist the intravascular pressure. This accident is rare, and usually occurs in old people. In most cases of retinal hæmorrhages, degeneration of the vessel-walls has preceded the extravasation of blood. The weakness may be due to vascular degeneration produced by age or by toxins circulating in the blood-stream, or both. The toxic effect of the blood is most evident in pernicious anæmia, leukæmia, scurvy, purpura, phosphorus poisoning, etc. In more chronic form it occurs in nephritis, diabetes, syphilis, and old age.

The small hæmorrhages which are so commonly seen in cases of vascular disease due to nephritis, diabetes, etc., and in acute anæmias, are of two kinds. The commonest form is a flame-shaped hæmorrhage with frayed-out or striated edges. The striæ take the direction of the nerve-fibres of the retina, and these hæmorrhages are in the most superficial layers of the retina. They are most often close beside the larger vessels, but may occur in any situation. Other small hæmorrhages are circular, with rather indefinite edges. They are situated in the deeper layers of the retina, and are less often seen than the superficial, probably owing to their deeper position.

The most striking form of so-called hæmorrhagic retinitis is seen in **thrombosis of the central vein of the retina** or of one of its branches. In these cases the patients are nearly always old, and have vascular disease, generally caused by chronic nephritis. Obscuration of vision is noticed, almost invariably on waking in the morning. The low blood-pressure and sluggish stream during sleep allow thrombosis to occur in a vessel the lumen of which is already narrowed by endovascular changes. When the central vein itself is involved, the obstruction is always immediately behind the lamina cribrosa, where the vessel is normally somewhat constricted. Thrombosis may, however, occur from local inflammation in

the orbit, such as orbital cellulitis by extension from facial erysipelas or from the nasal sinuses. On ophthalmoscopic examination, all the veins of the retina are found to be enormously dilated and very tortuous. The retina is covered with small hæmorrhages, which may be so closely packed in parts as to be confluent. The hæmorrhages are chiefly in the superficial layers of the retina, as shown by their flame-shape and striated appearance, but round and more deeply situated hæmorrhages are present. The whole retina is swollen and œdematous. In some cases there are spots and patches of whitish exudates. There may be some perception of light at first, but the retina soon ceases to perform its function and eventually becomes atrophied. When only a branch of the vein is affected the signs are confined to the area drained by the vessel.

Almost invariably in thrombosis of the central vein there are all the signs of degeneration of the vessel-walls. The normal light-reflex from the walls is usually bright and broad ("silver-wire" arteries). The arteries lose their translucency, so that where they cross veins the blood in the latter cannot be seen, as it usually can in normal vessels. There is evidence of undue pressure by the artery on the vein at the crossing, shown by distension on the distal side and constriction on the proximal side. The vein appears also to be deflected at the crossing, as if pushed aside. The veins show irregularity of contour, being unduly distended in some parts and constricted in others. The normally invisible walls become evident as white lines bounding the blood-stream, which may, indeed, be completely obscured in places. These changes need not affect all the vessels, but are patchy in their distribution.

**Treatment** is of no avail in thrombosis of the central vein, but the condition should be regarded as a danger signal and attention be directed to the primary cause, e.g. nephritis. Not infrequently the blind eye, after an interval of weeks or months, becomes painful owing to secondary glaucoma. In these cases it should be excised.

In **preretinal or subhyaloid hæmorrhage** the blood is extravasated between the retina and the hyaloid membrane of the vitreous, in the neighbourhood of the macula. Vision is suddenly obscured, and on examination a large round hæmorrhage is seen at the posterior pole of the eye. It soon changes shape, becoming hemispherical, with the upper border straight. This is due to gravity. Oc-

## RETINA, AFFECTIONS OF

asionally two such hæmorrhages are present simultaneously in the same eye. In time the upper layers become paler, probably owing to the red corpuscles gravitating downwards. The retinal vessels lie under the blood and cannot be seen.

The blood gradually becomes absorbed, leaving patches here and there in the affected area. Even after it has completely disappeared the area is usually studded with glistening cholesterol crystals. Vision is restored, but the condition may recur, and may be complicated by other vascular disorders.

In some cases of retinal hæmorrhage the blood bursts through the hyaloid membrane and becomes extravasated into the vitreous. The source of the blood in many vitreous hæmorrhages is uncertain, since it may come from the ciliary body. Such cases are the recurrent vitreous hæmorrhages in young adults. Blood in the vitreous usually becomes absorbed, leaving little trace save a few vitreous opacities. If, however, there is some general toxic condition, such as syphilis or nephritis, or if the hæmorrhages are frequently repeated, greater or less organization may take place, and the condition known as **retinitis proliferans** is produced. Here strands, films, or masses of fibrous tissue stretch into the vitreous, carrying new-formed blood-vessels upon them. They nearly always spring from the neighbourhood of the disc. Vision is much impaired, and may be abolished by detachment of the retina, which is a common sequel.

In other cases blood becomes extravasated between the retina and choroid, leading to so-called **exudative retinitis** or **massive exudation in the retina**. Ophthalmoscopically there is a large raised yellowish-white mass, resembling conglomerate tubercle. The vessels show degenerative changes, and there may be arterio-venous anastomosis, with enormous dilatation of the veins. Vision is eventually lost by detachment of the retina, glaucoma, or cataract.

Retinal hæmorrhage in the macular region is the cause of the condition known as **retinitis circinata**. It generally occurs in elderly women. There is a ring of bright white patches, with crenated edges, forming a circle or ellipse, sometimes horseshoe-shaped and open towards the temporal side, around the macula, following the course of the larger macular branches of the superior and inferior temporal vessels. The patches are at a deeper level than the vessels. The disease is unilateral in about

half the cases, and exudative retinitis has been seen in the other eye. The patches may disappear, with improvement of central vision.

### 3. EMBOLISM OF THE CENTRAL ARTERY OF THE RETINA

This condition causes sudden and complete blindness in most cases. Rarely, when there is a cilio-retinal artery present, some central vision may persist. Or only a branch of the central artery may be affected, in which case there is an indefinite scotoma corresponding to the area of retina supplied by the blocked vessel, settling down eventually into a permanent sector-shaped defect. The left eye is more frequently attacked than the right, owing to well-known anatomical facts.

The ophthalmoscopic picture is very characteristic. There is a large milky-white area occupying the macular region and extending around the disc, the outlines of which are indistinct. This is usually attributed to œdema of the retina. In the centre of this area, at the fovea centralis, there is a "cherry-red" spot, very conspicuous owing to the contrast with the background. It is due to the thinness of the retina in this spot allowing the red choroidal reflex to be seen.

Owing to the obstruction to the flow of blood the arteries are reduced to threads, and the smaller branches are invisible. The veins are not much altered, except that they are contracted on the disc. Sometimes spontaneously or by slight pressure on the eyeball the column of blood usually in the veins becomes broken up into bead-like chains. The beads move in jerks, sometimes in the normal direction of the blood-flow, in others in the reverse direction.

The œdema or coagulation necrosis gives place in the course of weeks to a transparent but atrophic retina. The vessels remain contracted and the disc becomes atrophied; macular pigmentation may supervene.

The pupil is dilated and does not react to light. In some cases perception of light is restored in the extreme temporal region of the field.

The above description applies to true embolism, and is generally associated with endocarditis, and especially mitral stenosis. Far more commonly, however, the condition is due to thrombosis following endarteritis, and associated with general arterio-sclerosis accompanying nephritis, etc. In these cases the onset is usually less sudden, and there may have been premonitory obscurations of vision.

## RETINA, AFFECTIONS OF

Cases of sudden blindness, with the same ophthalmoscopic appearances, followed by rapid and complete recovery, are to be attributed to spasm of the walls of the retinal arteries.

**Treatment** is only of avail in the earliest stages, and then only in cases of spasm of the arteries or true embolism. Inhalation of amyl nitrite, massage of the eyeball, and paracentesis have been employed in order to dilate the retinal arteries and to facilitate the passage of an obstructing embolus onwards into a less important branch of the artery.

### 4. RETINITIS PIGMENTOSA

This form of retinitis is so called from the deposits of black retinal pigment which are seen on ophthalmoscopic examination. The patient complains of being unable to see in the dusk (night-blindness). Central vision, as tested by Snellen's types, may be quite normal, but the field of vision is affected. In the early stages there is a ring scotoma; this very slowly but persistently increases in width until peripheral vision is abolished, and only a small field round the fixation point remains. In this stage patients are unable to get about, since they fail to see objects slightly removed from the direct line of vision (telescopic vision). At the same time they may be able to read small print and do fine work. At a later stage central vision begins to fail, usually owing to cataractous changes in the posterior part of the lens (posterior polar cataract). Eventually, after many years, the patient becomes blind.

The ophthalmoscopic picture is almost pathognomonic. The most striking feature is the multitude of jet-black spots, shaped like bone-corpuscles or spiders, scattered over the peripheral parts of the retina in each eye. Careful examination in the early stages will show that the extreme periphery is free from these pigment spots, so that the affected area is a zone near the equator of the globe. Soon the whole periphery is involved and the zone of pigment gradually approaches the central area. Only in the latest stages is the macular region affected, and usually cataractous changes supervene before this can be observed with the ophthalmoscope. Besides the isolated spots, pigment becomes deposited here and there in the perivascular sheaths of the veins. Owing to the migration of retinal pigment the choroidal vessels are unusually visible. The retinal vessels, both arteries and veins, become gradually smaller and eventually thread-like. Mean-

time the nerve-fibres degenerate and the disc shows atrophic changes; it becomes pale and waxy, but seldom chalky-white.

Several members of a family are often affected with retinitis pigmentosa, and not infrequently the parents are cousins. Insanity, idiocy, and other nervous diseases are common in such families, and in 3 or 4 per cent. of the cases the patients are deaf or deaf and dumb.

**Diagnosis.**—*Syphilitic retino-choroiditis* may simulate retinitis pigmentosa both in symptoms and in ophthalmoscopic signs. It is rarely, however, so symmetrical; generally the areas affected are limited to sharply defined patches in one or both retinæ. The Wassermann test and the absence of the characteristic individual and family history help to distinguish the two conditions.

Retinitis pigmentosa is primarily a degenerative condition of the choroid, specially affecting the blood-vessels. The nutrition of the retina suffers, involving first the rods (thus leading to night-blindness) and subsequently the nerve-cells and -fibres. The cause is doubtless some inherited tendency, but is at present obscure.

No **treatment** appears to have much influence on the progress of the disease. Strychnine is usually given, and electrical treatment seems to do good in some cases. It is permissible to extract the lens in the later stages, and temporary improvement of vision is sometimes obtained by this means. Anterior sclerectomy with the trephine has been done, but is of doubtful utility.

### 5. DETACHMENT OF THE RETINA

The retina may become separated from the choroid by being pushed up by a subretinal hæmorrhage or a sarcoma of the choroid, or by being pulled up by the contraction of organized exudates resulting from cyclitis. In many cases neither of these causes is evident, and the detachment appears to be spontaneous; in some of these cases there is a definite history of a severe blow upon the eye, in others there is a high degree of myopia, but in a considerable number no cause can be assigned. In some of these last cases detachment occurs spontaneously in the second eye after a longer or shorter interval.

Detachments of the retina which are not due to sarcoma of the choroid are conveniently grouped under the designation **simple detachment**, which is usually in the lowest part of the fundus, and, even if it does not commence in this situation, tends to be transferred there

## RETINA, AFFECTIONS OF

by the gravitation of the subretinal fluid. All detachments tend in time to involve the whole of the retina, resulting in complete blindness.

The first sign is usually cloudiness of vision, the detached area causing a positive scotoma. Parts of objects are obscured, usually the upper parts. If the detachment is shallow the scotoma may be only partial, the retina receiving sufficient nourishment from the subretinal fluid. The macula usually escapes at first, so that central vision is relatively little impaired. The earliest symptom may be spontaneous flashes of light (*photopsiæ*), especially in myopic cases, but these may occur without any detachment following. The tension is usually normal or diminished.

**Sarcoma of the choroid** may begin at any part of the fundus. If the macula is early involved, objects appear to be distorted (*metamorphopsia*, *micropsia*, etc.), and there may be a relative central scotoma for colours. A small detachment due to a sarcoma of the choroid may be accompanied by a large simple detachment in the usual situation below. In the later stages of the sarcoma the tension is raised.

Ophthalmoscopic examination should always begin with the mirror alone, the distant direct method. In cases of detachment there is nearly always some difference in the colour of the reflex from the pupil when the eye is moved in different directions. This arrests attention and suggests a detachment, which may otherwise be overlooked. The detached retina has a different tint from the normal fundus, though the difference may be slight if the detachment is shallow. In a typical detachment the retina is white or grey, with numerous folds, which may shift and oscillate when the eye is moved. The retinal vessels look nearly black, appear smaller than normal, and show a curve corresponding to every fold. With the direct method the displacement forward of the vessels can be measured, as compared with vessels in the still unseparated retina. In extensive detachments there are large balloon-like protrusions. Not infrequently a hole can be seen in the retina, through which the bright-red choroid is visible.

Part of the detachment is usually rounded and may have masses of pigment upon it. In some cases the vessels of the growth, differing from retinal vessels in that they anastomose freely, can be made out. This sign is pathognomonic.

The **prognosis** for vision is bad in all cases of detachment. Some cases of simple detachment

become cured spontaneously or after treatment, but the cure is rarely permanent.

**Treatment.**—In cases of sarcoma the eye should be excised at once. In simple detachment many methods are usually tried, with little success. If seen early the patient should lie immobile on the back for five or six weeks. Atropine is instilled, and the eye lightly but firmly bandaged. Mercury unctions and iodides by the mouth may be given.

Occasionally good results have followed scleral puncture over the site of the detachment with a Graefe knife or galvano-cautery.

### 6. GLIOMA OF THE RETINA

Glioma or neuro-epithelioma of the retina is a malignant disease of the retina occurring only in infants. It differs from all other malignant growths in origin and structure. It occurs as an independent growth in each eye in nearly one-fourth of the cases, and may affect several members of the same family.

The child, usually a baby, and nearly always less than 5 or 6 years of age, is noticed to have a yellowish reflex from the pupil of one eye ("amaurotic cat's eye"). On examination with the ophthalmoscope a yellowish-white mass is seen in the retina. Around the larger mass are numerous satellites, and polypoid protrusions may stretch forward into the vitreous (*glioma endophytum*). When the extension is chiefly in the deeper parts of the retina (*glioma exophytum*) the appearance is that of a detached retina. There are often small hæmorrhages upon the surface of the growth.

If untreated, the growth extends until it fills the eye, leading to rise of intraocular tension. There is severe pain until the growth bursts through the walls of the globe, usually just outside the margin of the cornea. The tumour then grows very rapidly, forming eventually a huge fungating mass. Metastasis occurs in the preauricular gland and cranial and other bones, less commonly in other organs. The optic nerve is early affected by direct extension. The preglaucomatous stage lasts from six to twelve months.

The chief conditions (*pseudogliomata*) which may be mistaken for glioma are: (1) inflammatory deposits in the vitreous, with or without detachment of the retina; (2) tubercle of the choroid, especially the confluent type; (3) persistence of the posterior part of the foetal fibro-vascular sheath of the lens. Cases of the first group are the commonest, and are due to a quiet form of cyclitis, usually caused by post-

## RETROVERTED GRAVID UTERUS

basic meningitis or an acute specific fever. There are usually signs of iritis—posterior synechiae, irregularity of the pupil, retraction of the base of the iris, etc. The tension is usually subnormal. In all cases of doubt the pupils should be dilated and both eyes examined with the ophthalmoscope under a general anæsthetic; only under these conditions can the tension be satisfactorily determined.

**Prognosis and treatment.**—Glioma of the retina invariably causes the death of the patient unless the eye is removed at an early stage. Care should be taken to remove as much as possible of the optic nerve, which is early involved. The prognosis as to life is good if there is no recurrence in the orbit within three years, but the other eye should be examined periodically for still longer. Life may be saved in bilateral cases by early removal of both eyes.

Pseudoglioma always leads to partial or total blindness, and the eye often shrinks. It cannot always be diagnosed from glioma with certainty, and seeing that the life of the patient is at stake, the eye should be excised.

If the cut end of the optic nerve is found to be infiltrated with glioma on microscopic examination, or if recurrence has taken place in the orbit, the orbital contents should be exenterated.

J. HERBERT PARSONS.

**RETINOSCOPY** (*see* EYE, EXAMINATION OF).

**RETROBULBAR NEURITIS** (*see* OPTIC NEURITIS AND NEURO-RETINITIS).

**RETROFLEXION AND RETROVERSION OF UTERUS** (*see* PELVIC ORGANS, FEMALE, DISPLACEMENTS OF).

**RETROPERITONEAL TUMOURS** (*see* ABDOMINAL TUMOURS, DIAGNOSIS OF).

**RETROPHARYNGEAL ABSCESS** (*see* ABSCESS, RETROPHARYNGEAL).

### RETROVERTED GRAVID UTERUS.

—Retroversion and flexion of the gravid uterus is a complication which gives rise to symptoms about the end of the third month of pregnancy if the fundus of the uterus is prevented by the promontory of the sacrum from rising above the plane of the pelvic brim. In many cases spontaneous correction of the displacement occurs before symptoms arise, or there may be a little irritation of the bladder. If, however, the fundus remains caught up

under the sacral promontory, the uterus is said to be “incarcerated,” and bladder symptoms supervene until retention of urine results, this being in many cases the first symptom to attract the patient’s attention. The condition is much commoner in multiparæ than in primigravidæ, owing to the fact that, in the former, retroversion with flexion of the uterus is more common.

Apart from the bladder symptoms which commonly occur, retroversion is liable to cause abortion; further, it is sometimes found in cases of severe vomiting of pregnancy.

**Diagnosis.**—This is suggested by the symptoms, and is confirmed by physical examination. On abdominal examination a full bladder is felt, rising in some cases to the umbilicus; this is confirmed by passing a catheter. The bladder being emptied, the fundus of the uterus cannot be felt at the plane of the pelvic brim. On vaginal examination, the cervix is felt very far forward, being drawn up in some cases almost out of reach, behind and above the back of the symphysis pubis. Bimanually, the body of the uterus is not felt in the anterior fornix in front of the cervix, but can be made out behind the cervix in Douglas’s pouch.

The only condition which is likely to be confused with this is *ectopic pregnancy with pelvic hæmatocele*. In the latter, however, pain will be a prominent symptom with some bleeding; bladder symptoms will not be as evident; the tumour is less definite and solid in consistence than the pregnant uterus, and the body of the uterus, not much larger than normal, can be felt in front of the swelling. In rare instances a *fibroid* or an *ovarian cyst* in the pelvis complicating pregnancy may cause retention of urine.

**Treatment.** The general lines of treatment are correction of the displacement, and the insertion of a pessary for a few weeks until the uterus has risen out of the pelvis. If the condition has been discovered before retention has occurred, correction of the displacement can be carried out by the usual methods of pulling on the cervix with a volsella and pushing up the body of the uterus with two fingers in the vagina or rectum, aiding this manœuvre by raising the pelvis. These manipulations should be carried out very gently, and if any difficulty is experienced an anæsthetic had better be administered.

If retention of urine has occurred, the patient must be put to bed for a few days and catheterized eight-hourly. If she be kept prone

## RHEUMATISM, ACUTE

after the catheter has been passed, the uterus sometimes drops forward and spontaneous correction of the displacement occurs. If not, it is best to correct the position of the uterus by manipulation under an anæsthetic. After this a pessary is inserted for three or four weeks to prevent the uterus from dropping back.

As regards the bladder, a certain amount of weakness generally supervenes for some little time. The patient should be encouraged to pass her water naturally, after which a catheter is used to evacuate any residual urine that remains; when there is none, the patient can be considered cured. During catheterization a course of hexamine must be given.

GORDON LUKER.

### RHEUMATISM, ACUTE. *Etiology.*—

Acute rheumatism is still classified in treatises among the diseases of doubtful etiology, but in the last twenty years the problem has advanced a definite stage and it is now generally recognized to be an infection.

Some hold that there is probably more than one variety of infection; others, that its nature is unknown; others, the writer included, believe the cause to be a diplococcus belonging to the large family of streptococci. The diplococcus in question has been isolated from the cardinal lesions of the disease both during life and after death, has been grown in pure culture, and has reproduced these lesions in rabbits and monkeys, from the diseased tissues of which in turn it has been isolated in pure culture.

**Predisposing causes.** *Heredity.*—Among important causes predisposing to this infection, heredity is strongly in evidence, being noted in about 58 per cent. of the cases in the private records of the late W. B. Cheadle. Hereditary cases must be distinguished from the rare examples of congenital rheumatism in which a mother suffering in pregnancy from the acute disease infects the foetus through the placental circulation.

*Age.*—Acute rheumatism is uncommon under the age of 5, increases in frequency up to puberty, and then gradually diminishes.

*Sex.*—It attacks more female than male children, in the proportion of about three to two.

*Climate.*—Although ubiquitous, the disease is most frequent and severe in temperate and changeable climates, particularly in autumn and spring. A disease of large cities, the incidence is favoured by cold winds and damp.

I attribute importance to the dust of large towns, and find that a long spell of dry weather followed by cold winds is particularly dangerous. I also attach much importance to cold, damp surroundings and houses, in the belief, not that these are causes of the disease, but that they favour its incidence by lowering vitality and are potent factors in producing the chronic and obstinate forms of the complaint. In many instances where the claims of pyorrhœa have been put forward I would lay more stress on the fact that such patients have come from riverside localities or from poor houses which are damp and built on cold clay. There is, in fact, a fairly clear clinical distinction between cases of acute rheumatism due to virulent infection and those the result of an attack not originally of great violence, but aggravated by cold and damp. The latter group tends to develop fibrositis and arthritis of an inveterate type, the former to develop the severer forms of carditis.

*Epidemics.*—There is evidence that waves of increased frequency, sometimes affecting quite a localized area, may occur. Striking examples which point to contagion are on record, but in comparison with the frequency of the disease are exceptional.

Other infections, such as scarlet fever, measles, influenza, and diphtheria—diseases which cause tonsillar inflammation—may pave the way for a secondary invasion by the rheumatic germ. This fact, in cases other than scarlet fever, is perhaps not appreciated so fully as it deserves to be.

**Pathology.**—The changes in the tissues produced by the infection are remarkably uniform. Our knowledge of the essential pathology is deficient owing to our ignorance of the nature of the rheumatic toxins, but the gross tissue changes are well recognized.

The various lesions may be regarded as due to local infections and the rheumatic nodule taken as the type. In this we find three stages of tissue change; the outer zone showing congested blood-vessels and swollen connective tissue, the middle zone showing a leucocytic infiltration, and the innermost a small area of necrosis. A fortunate section of a nodule cut in an early stage will show the diplococci, generally on the outer edge of the necrotic area.

All the cardinal lesions own the same features, namely, vascular dilatation, swelling of the connective tissues, leucocytic infiltration and necrosis, but the changes produced are modi-



## RHEUMATISM, ACUTE

fied by the functions of the affected part. Thus, the extremely vascular synovial membranes of the joints, exposed as they are to injury from pressure, resemble in severe cases crimson velvet in appearance, and many minute blood-vessels may rupture in them. The heart-wall with its composite structure of connective tissue and muscle shows two types of injury—that to the connective tissue and its blood-vessels and nerves, and that to the muscle. Fatty changes can readily be recognised in the muscle, and are due to toxins.

The effusions, whether pericardial, synovial, or pleural, vary in character with the virulence of the infection. Thus, in rheumatic pericarditis, if the infection is highly virulent the effusion is blood-stained, if less severe in the earliest stage it is serous, and if more obstinate, sero-fibrinous. It is generally recognized that, whatever the exact nature of the rheumatic toxins, they do not, however deadly, produce pus.

In the brain it is more difficult to discern the characteristics of the rheumatic lesions, but we find the usual type of connective-tissue change in the pia mater, and, as elsewhere, a tendency to perivascular exudation around the minute blood-capillaries. The nerve-cells show chromatolysis and eccentric position of the nuclei, such as have been recorded in other cerebral infections and toxæmias.

Much attention has been given to the sub-miliary nodules which are a feature of the connective-tissue changes in rheumatism. These I look upon as representing a phase in the tissue reaction. They consist of fusiform areas formed by large spindle-shaped cells, often multinuclear, around which lie plasma cells and leucocytes, and are slowly replaced by connective tissue. They have been much studied in the heart-wall by Aschoff, Coombs, and others, and are of interest in this situation because of their relation to the modern researches on disturbances of the cardiac rhythm as the result of injury to the A.V. bundle.

Thus, in the pathology of acute rheumatism we see multiple foci of infection, rapid destruction of the diplococci by leucocytes and connective-tissue cells, and the formation of scar tissue where there has been necrosis.

It would be an error to suppose that the tissue reaction to the infection always follow the same course. Three main types may be distinguished: (1) the acute, well exemplified by acute rheumatic arthritis; (2) the fibroid,

illustrated by mitral stenosis and chronic sclerosing pericarditis; and (3) the malignant type, in which the diplococci are not destroyed by the tissues, illustrated most clearly by rheumatic malignant endocarditis. There may also be imperfect healing, with the resulting danger of survival of the infection in the diseased areas. When once the general principles of the pathology of this disease are understood clearly, we can explain with ease its many symptoms, and need not exalt to a separate entity a fibrositis occurring in the rheumatic, nor invariably postulate a superimposed infection when malignant endocarditis develops.

**Symptomatology and clinical course in childhood.**—It has been the custom to give a description of acute rheumatism as it occurs in adult life and to draw clinical distinctions between these symptoms and those met with in childhood. Its nature is, however, more correctly presented by describing its course in childhood and then laying emphasis on the changes in its behaviour when it occurs in the adult.

In childhood, then, the lesions are widespread. Though all are not yet well known, some occur so frequently that they have been termed "manifestations" of the disease. At the onset of the illness several of these manifestations often appear together; in severe cases nearly all of them may be seen within a short period.

Thus, we often find a sore throat associated with arthritic pains and endocarditis, and chorea with heart disease. In a severe case an erythema, rapid anæmia, nodules, and pleurisy may be added. On the other hand, one organ may be singled out for attack, almost to the exclusion of others; for example, the heart; or the brain in cases of repeated chorea. On close inquiry it is generally discovered that the predominant lesion is not quite solitary, the other organs being affected so lightly in comparison that the symptoms are easily overlooked.

In very severe cases, diarrhoea and vomiting with great muscular weakness may occur. The symptoms will then probably develop along particular lines depending upon the organs most affected.

The cases which commence insidiously may show various indeterminate symptoms, such as lassitude, anæmia, persistent but slight fever, headache, fleeting pains in the tendons, muscles, and epigastric region, epistaxis, irritability of temper, and nervousness. It is only in very

## RHEUMATISM, ACUTE

exceptional instances that subcutaneous nodules are an early and prominent symptom.

The course of the illness is necessarily very variable, because so much depends upon which organs are most severely attacked, and upon the number of manifestations.

In childhood we should not regard acute rheumatism as an infection running a definite course as typhoid fever does, but realize that it may extend, with remissions, over many months. Thus, an attack of arthritis and carditis may be followed by chorea, and nodules may then appear, and possibly a fleeting erythema. Exceptionally a case may prove fatal after eighteen months or more of continued rheumatic activity; there may be no evidence of fresh infection, and we are left with the conviction that the child had never recovered from the original attack. Emphasis has been laid upon these points in the history of acute rheumatism because they explain the great difficulties that arise in attempting to give an adequate account of the clinical symptoms. A knowledge of them also puts us on our guard against accepting the view that an absence of a history of arthritis is evidence against the rheumatic origin of obvious heart disease or chorea in a particular case.

Children seldom succumb to their first attack. The two great dangers of the disease are—(1) the tendency to repeated attacks, and consequent liability to death from renewed assaults upon the heart; and (2) the damage done to the mechanism of the heart by scar-formation, which in later years often results in death from chronic morbus cordis. Though, in proportion to the incidence of the disease, death is rare in a first attack, this event may certainly occur, particularly in very young children with a strong hereditary tendency.

The most important manifestations of acute rheumatism in childhood are—

1. Carditis.
2. Arthritis, fibrositis, and teno-synovitis.
3. Chorea.
4. Sore throat.
5. Cutaneous manifestations, such as erythematous and purpura.
6. Subcutaneous nodules.
7. Pleurisy and respiratory affections.
8. Progressive anaemia.
9. Hyperpyrexia, which is rare in this country.

**1. Cardio-vascular manifestations** (*see also* ENDOCARDITIS; MYOCARDITIS; and PERICARDITIS).—The rate at which these lesions develop

varies greatly. Their onset may be so gradual and lacking in symptoms that examination of the heart with care in all cases of suspected rheumatism remains a cardinal rule. Rare lesions are *arteritis* and *phlebitis with thrombosis*. The latter sometimes implicates the superior vena cava and its tributaries, at other times the veins of the lower extremities.

**2. Arthritis.**—This is seldom severe in childhood. The larger joints are usually affected, but the articulations of the fingers and toes are not uncommonly attacked at this age, and a condition resembling acute rheumatoid arthritis with wasting of the muscles may result. Occasionally an acute swelling of the metatarsophalangeal joint of the great toe simulating a gouty arthritis is met with. *Lumbago* and *fibrositis* in other muscles, as for example the cervical, may occur in childhood, and *teno-synovitis*, particularly in the hamstring muscles, is frequent. There may be much pain with remarkably little articular swelling. The arthritic manifestations subside rapidly, as a rule, but the pain connected with fibrositis may prove obstinate, even in the young.

**3. Chorea** may occur at any period in a rheumatic attack and, in girls, is often for several years the sole predominant lesion. It may even remain the only prominent manifestation of the disease. Its onset may be very gradual, or acute and even abrupt, and the attacks may be of all grades of severity. The mild cases are often protracted, while the acute cases may terminate rapidly. Six weeks is frequently given as the usual duration of an attack, but this is a favourable estimate so far as London children are concerned. Other nervous disturbances must not be overlooked in rheumatism, such as undue nervousness and emotional disturbance, persistent headache and mental change. Symptoms suggestive of peripheral neuritis are occasionally met with in attacks of chorea.

**4. Sore throat.**—The throat may be red and glazed, or there may be deposits between the follicles or a membranous exudation. The cervical glands are frequently enlarged, and adenoids or catarrhal rhinitis may complicate the tonsillar condition.

**5. Cutaneous manifestations.**—*Erythema marginatum* and *erythema papulatum* are the most frequent. These may appear as solitary lesions or, more frequently, during an attack in company with other manifestations. *Erythema marginatum* appears as rings of a pink colour, slightly raised, and varying much in size and

## RHEUMATISM, ACUTE

distribution. With its onset there may be irritation or tingling sensations, and a stain is left by the fading rash. In the papular form the papules come out in crops, vary in size, are generally situated on the dorsal surfaces of the extremities, and leave a stain in fading. *Purpura* occurs either in connexion with the erythemata or alone. It may be very severe, may take the form of numerous small macules scattered over the lower extremities, or may produce an irregular tracery over the legs caused by interlacing lines of hæmorrhage. *Bullous eruptions* are much less frequent. *Psoriasis* appears to me to be an occasional result of rheumatism. *Erythema nodosum*, though looked upon by some as tuberculous and by others as a special disease, is undoubtedly closely allied to acute rheumatism in some instances, if not a direct result of that infection.

6. **Subcutaneous nodules.**—These lesions, which are inflammatory exudations and not fibrous nodules, appear over bony prominences and along tendons. They are met with on the back of the head, around the elbow-joints, on the patellæ, over the vertebral spines, on the ankles, and less frequently on the crests of the ilia and the spines of the scapulæ; they may even occur on the malar bones. Common sites are the extensor tendons of the fingers and toes. The nodules vary in size from a pin's head to a small marble, and are often best detected by sight, when the light is thrown obliquely and the skin gently stretched. They are painless, and can be moved under the skin when gently pressed upon; sometimes they are abundant; they usually disappear entirely, although the larger ones may take months in the process. Very occasionally they are the most obvious manifestation, but as a rule they would be of no practical importance were it not that they are a warning of a type of rheumatism in which the heart is particularly liable to suffer. This fact makes them one of the most interesting and important manifestations of the disease, and one almost confined to childhood.

7. **Respiratory manifestations.**—*Pleurisy* frequently occurs when there is pericarditis, and is sero-fibrinous in character. When the inflammation affects the pleuræ where they overlap the pericardium, pleuro-pericardial friction may be heard. This sign must be distinguished from pericardial friction by its respiratory and cardiac rhythms; its import is less serious. The frequency with which rheumatic pleurisy occurs alone is difficult to estimate. In ex-

ceptional cases the effusion may be sufficient to require paracentesis, and the diplococcus has been isolated from the exudation. There is no unusual feature in the pleurisy, but it is a painful complication in carditis.

*Pneumonia*, broncho-pneumonic in distribution, is one cause of an unexpectedly high temperature in acute rheumatism apart from hyperpyrexia, and may be overlooked from a desire not to disturb the patient. *Acute pulmonary œdema* is a rare but dangerous condition. The physical signs are characteristic, for fine crepitations appear in the upper lobes of the lungs and are accompanied by great respiratory embarrassment.

Hirsch and others have described rheumatism of the crico-arytenoid joints with accompanying laryngitis.

8. **Anæmia.**—Few diseases produce a more rapidly progressive anæmia. During the active phase the red blood-corpuscles fall quickly in number, and recovery from this condition may be very slow. The hæmoglobin is also diminished. There may be a moderate leucocytosis or, more rarely, one which is considerable. In the virulent form of the disease severe hæmolysis may occur.

9. **Hyperpyrexia** (cerebral rheumatism).—This formidable complication usually occurs in the third or fourth week of the disease, though it may supervene after the temperature has been normal for days. Headache, sleeplessness, vomiting, and delirium are convincing signs, and frequency of micturition and cutaneous hyperæsthesia have occasionally been recorded. The temperature may rush up with a bound, or rise slowly at first and then leap to 107° F., or even to 110° F. The patient becomes semiconscious and the breathing irregular. There are spasmodic twitchings, the pupils are contracted, and the face is livid. The other signs of active rheumatism may abate during the hyperpyrexia. The condition is one of extreme danger, even when the treatment is prompt.

Fortunately, not all the cases of hyperpyrexia are of this severe type; in milder examples, though the cerebral symptoms are prominent, the temperature does not rise beyond 105° F. Several minor attacks may occur in the course of a single attack of acute rheumatism. It is unusual to find any gross lesion after death.

Besides the manifestations of the disease in childhood described above, the following may be mentioned:—

## RHEUMATISM, ACUTE

10. **Alimentary disturbances.**—Rheumatic children are liable to attacks of vomiting and fever accompanied by the passage of pale stools, and also to mucous colitis. In adults acute dilatation of the stomach and painful distension of the colon may cause serious symptoms, particularly if associated with carditis.

Peritonitis is a rare event. I incline to the view that acute rheumatism is a cause of appendicitis.

11. **Acute nephritis** may result from the rheumatic infection, and it is worth considering whether the chronic infection may not cause interstitial nephritis, for granular kidney is frequently met with in women suffering from advanced mitral stenosis.

12. **Mastitis.**—Mastitis occurs in rheumatic female children between the ages of 10 and 13 years, but whether rheumatic in origin is not yet proven.

13. **Thyroiditis** has been described, and in chorea some swelling of the thyroid gland is occasionally noticed. The association of rheumatism with Graves's disease has been long recognized.

14. **Ocular manifestations** are rare, but acute irido-cyclitis has been recorded in cases which were not gonorrhoeal in nature. Conjunctivitis occurs more frequently.

15. **Sialodochitis fibrinosa.**—I have observed recurrent parotid swellings during acute rheumatism.

**Symptomatology of acute rheumatism in the adult.**—If we follow the story of a child, subject to attacks of acute rheumatism, into adult life we often find that the tendency to acute attacks diminishes and the manifestations decrease in number, while otherwise the character of the disease alters but little. On the other hand, we also meet with acute first attacks of what is often termed *articular rheumatism* or *rheumatic fever* in the adult.

The illness commences with a chill and migratory pains in the joints and muscles. Some passing soreness of the throat is frequent, and the temperature is raised. Then one or more of the larger joints become swollen. There may be an erythematous flush over them; the synovial cavities become filled with fluid and the periarticular tissues swollen. The tendons around these joints are often implicated. Pain is intense and even agonizing, but no suppuration occurs. The onset of inflammation is rapid, and the subsidence in any joint may be equally abrupt, but the arthritis usually flies from joint to joint. There is general malaise,

and the tongue is thickly coated with a white fur. The pulse is febrile, full and bounding. A striking symptom in this group of cases, but one which is sometimes also met with in young children, is profuse sour sweating with an eruption of sudamina. The temperature, though raised, follows no definite course, the duration of the pyrexia varying on the average from two to four weeks. The urine is febrile, the urea and uric acid being increased and the colour deep. Albumin may appear in small quantities. Anæmia results from a severe attack, and there is an increase in the fibrin of the blood. There may be acute cardiac dilatation, mitral endocarditis, or, in grave cases, a pancarditis. Pleurisy and pneumonia may occur, and in rare instances hyperpyrexia. In a favourable case the fever generally subsides in three weeks, leaving the patient with stiffened muscles and weak joints, which usually respond rapidly to appropriate treatment.

Unfortunately, the course of articular rheumatism may be greatly prolonged by relapses. These are more frequently met with in the sub-acute cases, and may prolong the illness for many weeks. The patient becomes intensely depressed, anæmic, and weakened by the pain and continued activity of the disease, and may finally succumb to active carditis. Death, however, is very unusual in acute articular rheumatism; when it occurs it is the result of a severe carditis or hyperpyrexia, or, very rarely, of acute dilatation of the stomach.

Recovery may be complete or be rendered imperfect by cardiac affections or by an unusual obstinacy in the arthritic lesions, so that troublesome stiffness and partial crippling result. In such cases complete restitution of health may be very slow.

The following are some clinical differences in the behaviour of acute rheumatism in the adult as compared with its behaviour in the child:—

1. The manifestations are less varied.
2. Heart disease is less frequent and severe.
3. Articular symptoms are more severe.
4. Sweating is more frequent.
5. Nervous symptoms, notably chorea, are less frequent.
6. Hyperpyrexia is probably more liable to occur.
7. Subcutaneous nodules are less frequent.
8. Malignant rheumatic endocarditis is more frequent.
9. Anæmia is less profound.
10. The tendency to drift into the rheumatic state is less prominent.

## RHEUMATISM, ACUTE

**Diagnosis in the child.**—The most urgently needed diagnosis is that between acute rheumatism and *osteo-myelitis*, for an error may cost a life. The onset of *osteo-myelitis* is usually more acute and the appearance of the patient more suggestive of sepsis. High fever, rigors, and delirium may occur. Pain is often extreme and limited to the region of the epiphyses rather than to the articulations, and the lesion is usually solitary, the limb becoming intensely inflamed and sometimes brawny.

**Acute poliomyelitis.**—At first sight there would seem to be no likelihood of confusion, yet experience proves that in cases in which pain is a prominent feature, the distinction is not always easy in the early stages. Loss of reflexes, rapid muscular wasting, and the absence of arthritic swelling and *morbus cordis* are valuable diagnostic points.

**Infantile scurvy.**—Acute rheumatism is very unusual in children under 2, and at that age scurvy should always be uppermost in the mind. The periosteal swellings, purple swollen gums, ecchymoses, hæmorrhages, and faulty diet serve to distinguish the two conditions. Treatment rapidly makes the position clear.

**Congenital syphilis.**—(1) Epiphysitis and periostitis, causing sometimes a pseudo-paralysis, occur in infancy, and the stigmata of syphilis will point to the correct diagnosis. (2) Syphilitic arthritis in older children is generally singularly painless, and often confined to the knee-joints. With it are often associated interstitial keratitis, peg teeth, and deafness. The result of the Wassermann reaction affords valuable evidence.

**Gonococcal arthritis** shows itself in the first five weeks of life and is associated with ophthalmia. Gonococci may be demonstrated in the effusions.

**Acute pneumococcal arthritis** is generally a complication of pneumonia and empyema, and may be suppurative.

**Acute tuberculous arthritis.**—The diagnosis may be extremely difficult, although the presence of tuberculous lesions elsewhere, the demonstration of tubercle bacilli in the arthritic exudation, and the absence of a history of acute rheumatism usually suffice to settle the question.

**Meningococcal arthritis** may be distinguished by the isolation of the micro-organism from the arthritis exudation, or from the cerebro-spinal fluid after lumbar puncture.

**Still's disease** usually commences before the second dentition. Its course is protracted, the

swelling is mostly periarticular, muscular wasting is conspicuous, and anæmia, pyrexia, and sweating are prominent. The lymphatic glands and spleen may be enlarged, and, if the heart is affected, it is by a terminal pericarditis. Salicylates have no influence on the temperature or course of the disease.

**Arthritis deformans** is comparatively rare, but remarkable examples do occur, with lipping and bony outgrowths, extreme muscular wasting, and absence of cardiac lesions.

**Hæmophilia** occasionally causes painful arthritis, by hæmorrhage into the synovial cavity. The abrupt onset and the history and sex of the patient usually lead to the correct diagnosis.

**Appendicitis** may be simulated by acute rheumatic arthritis of the right hip-joint. Freedom of the movements of the joint is a distinctive point.

**Diagnosis in the adult.** *Arthritis deformans.*—The symmetrical implication, particularly of the smaller joints, the rapid wasting, the failure of salicylates to relieve the pain, the persistent tachycardia without valvular disease, and the protracted course are among the distinguishing features, but there are exceptions to almost all these diagnostic indications, and it is probable that acute rheumatism may be one cause of arthritis deformans.

**Gout** occurs usually after 30 years of age. In a classical case the occurrence of tophi, the violent pain and great swelling and redness of the metatarso-phalangeal joint of the great toe, with the absence of endocarditis, are all important points. It is possible that acute rheumatism may precipitate a gouty attack in a patient in the gouty state.

**Gonococcal arthritis** often causes difficulty. The stubbornness of the arthritis, the frequency of iritis, and the painful plantar fasciæ are suggestive. A history of urethritis and prostatitis is of prime importance. The isolation of the gonococcus is conclusive.

**Pyæmic arthritis**, in both adults and children, may lead to error. Reliance must be placed on the history, the occurrence of high irregular fever, rigors, and prostration. Subcutaneous abscesses may form, and the heart, though rapid and feeble, is generally free from endocarditis. Withdrawal of fluid from an articulation will establish the diagnosis.

**Prognosis.**—The prognosis is concerned with the danger to life during an attack, the general outlook as to the future, and the liability to recurrences.

## RHEUMATISM, ACUTE

A first attack is not usually dangerous to life. The chief exceptions to this statement are furnished by the occurrence of hyperpyrexia, the mortality of which under the most careful treatment is nearly 50 per cent., and of acute carditis, which is especially dangerous in children under 6 years of age. When one manifestation follows another and the child drifts into a state of rheumatic toxæmia, the outlook is grave. In adults prolonged cases of a somewhat similar type may end fatally by cardiac failure, and in this connexion special mention must be made of dilatation of the stomach and colitis, which may add much to the gravity of the illness.

The younger the child, the greater the liability to cardiac complications, and in both child and adult it is the cardiac lesions which usually determine the future. The prognosis of these lesions is considered under **ENDOCARDITIS, ACUTE**; **MYOCARDITIS**; **PERICARDITIS**.

The patient's home surroundings greatly influence the prognosis. Poverty and damp and insanitary houses are serious factors.

The liability to recurrences depends in part upon the general surroundings, the history of a strong family tendency, and the age, but in all probability the character of the infection also exerts an important influence. It is difficult with our present knowledge to estimate this latter factor except by the actual course of the illness.

**Treatment. Preventive.**—The first consideration is that of prevention, for we have unfortunately no remedy that controls rheumatic heart disease.

Children with a family history of acute rheumatism should, if possible, be removed from unsuitable houses and surroundings. Attention should be paid to recurrent attacks of tonsillitis, and in such cases the tonsils should be enucleated. Parents and teachers in schools should be instructed in the nature and common manifestations of the disease. "Growing pains," emotional disturbances with irregular movements, headaches, lassitude, and shortness of breath on exertion are early symptoms which should receive immediate attention.

"Rheumatic" children need sympathetic handling. Nervous and easily tired, full of energy but with little reserve strength, they require considerably more rest than the normal child of the same age, and the tendency for the disease to develop in the late autumn and early spring points to particular care as to

their clothing and protection from inclement weather.

The greatest care should be taken to allow ample time for convalescence from a first attack.

**Management of the acute attack.**—In the case of an adult, helpless with a severe attack of acute articular rheumatism, strong nurses are very advisable. The sick-room should be airy but not draughty, the bed of a convenient height and not too wide. The patient, clothed in a light flannel night shirt, should lie between soft blankets on a firm mattress.

The *diet* should be fluid, consisting chiefly of milk diluted with water, soda water, or barley water. If necessary the milk may be peptonized or citrated (sod. cit. 20 gr. to 1 pint of milk). An adult will take about three or four pints of milk and water in the twenty-four hours, and, when he is thirsty, may sip home-made lemonade to which potassium bicarbonate (20 gr. to 1 pint) has been added. Stimulants are only needed for emergencies.

During convalescence children and most young adults can take light meals, such as fish, chicken, and eggs. I have no belief in the curtailment of these foods, unless there is clear indication of the presence of renal disease or the patient is elderly and with gouty tendencies.

**Medicinal and local treatment.**—The acute articular form is that which reacts well to the salicylates. When commencing the treatment the bowels should be well opened. At first calomel, followed by a saline, is generally indicated; afterwards milder aperients such as liquorice, senna, and cascara are sufficient.

For a strong adult the following mixture may be given every two hours for six doses, then every three hours for four doses, and then every four hours:—

17 Sod. salicyl. gr. xx.  
Sod. bicarb. gr. xv.  
Syr. zingib. ℥xx.  
Aq. chlor. ad ʒi.

Some prefer to use aspirin, but it should be given with plenty of water, for, when pushed, it is liable to upset the digestion.

In some cases the pain is so severe that it may be necessary to give, in addition to the salicylates, 10 gr. of Dover's powder, or liq. morphinæ hydrochloratis, or nopenal.

Local applications should be such as do not require frequent disturbance of the patient, and warm wool answers this purpose.

## RHEUMATISM, ACUTE

Fuller's alkaline-and-opium lotion can also be used ; the formula is :—

R̄ Pot. bicarb. ℥ss.  
Tr. opii ℥i.  
Glycer. ℥ii.  
Aq. ros. ad ℥xii.  
Fiat lotio. To be applied warm.

When dealing with an asthenic case it may be necessary to combine tincture of nuxvomica or sal-volatile with the salicylate of soda.

For a child of 10 years, an average dose of salicylate of soda is 10 gr., combined with bicarbonate of soda, 5 gr. I do not consider it necessary to give double the dose of bicarbonate of soda to that of the salicylate, as has been widely recommended. Salicin is the safest drug for very delicate children, given as a powder in milk in 10–20-gr. doses. Every practitioner who is inclined to push the salicylates, particularly in the case of children, should be well acquainted with the symptoms of their toxic action. Among the most important of these are deafness, giddiness, dimness of vision, and frequent micturition. Headache and obstinate vomiting, a slow and irregular pulse, with feeble cardiac sounds and cold, clammy extremities, are more severe signs. In rare cases death may occur, preceded by slow and deep breathing and coma. I have seen all these symptoms, and Langmead also has directed especial attention to these dangers.

When the case is running a favourable course the temperature falls and the pain subsides, but the salicylate treatment should be continued, in diminishing doses, for at least a fortnight after the temperature has reached the normal line.

Quinine is a useful drug during convalescence, and colloidal iodine in 3-min. doses may help to promote recovery of the swollen tissues, and is taken more easily than iodide of potassium. When there is much anæmia, iron and arsenic in alkaline solution are indicated, but with the adult there is need for caution in the use of iron, for there is considerable evidence that residual pain after the attack is liable to increase when the stronger acid preparations are used.

Massage is a valuable aid in restoring strength to the stiffened limbs before any attempt is made at walking, and there can be no doubt that baths and douches at a spa accelerate this phase of the recovery.

Many acute cases run a favourable course on

these lines of treatment, but others, especially those of a subacute type, continually relapse. The crux of the situation lies in the cause of these relapses. Some attribute them to chill, others to too lavish feeding, others again to premature exertion. I admit all as possible factors in particular cases, but believe the explanation lies in the nature of the infection, which, if the tissues do not respond actively, is characterized by waves of exacerbation, as the severe carditis of childhood illustrates.

No one, so far as I am aware, has discovered a method of destroying the diplococcus in the tissues, and the problem in these cases is how to rouse a patient's resistance to the infection. No one method has succeeded, and sometimes every attempt has proved a miserable failure. For a strong patient, sudden pressing of the salicylate treatment may succeed by giving relief from pain and consequently allowing sleep and rest. In other cases it is worse than useless, and for such the pain may require opiates, and the general prostration stimulants and tonics. The sick-room may be dull, dark, and cold, or a liquid diet may be persisted in over-long. A single joint may be the obstacle, and need careful splinting. In spite of all difficulties and risks, it may be necessary to move the patient to a more suitable locality or to a nursing home where radiant heat and cataphoretic treatment can be obtained, or to a hospital where more efficient nursing is available.

An English winter is better avoided after a severe attack of articular rheumatism. A sunny inland locality and a gravel soil are the most suitable.

**Cardiac rheumatism.**—The leading indications are :—

Immediate rest. The assistance of trained nurses is most valuable.

The relief of pain and distress.

The avoidance of all strain upon the heart.

Care in convalescence.

The chief external applications for relieving pain in pericarditis are the ice-bag, leeches, and hot applications, including hot wool. The drugs that are of most value are considered under ENDOCARDITIS, ACUTE; MYOCARDITIS; and PERICARDITIS. Convalescence is prolonged, and should be marked by steady forward progress and not by abrupt changes.

**Acute rheumatism with hyperpyrexia.**—The most successful method of treatment has been the use of the cold bath. The condition is urgent, the treatment anxious. The patient is

## RHEUMATISM, ACUTE

lowered into a bath of a temperature of 75° F., and to this blocks of ice are added. There is danger of collapse, and all must be in readiness for removal of the patient and for combating shock. The temperature must be taken frequently while in the bath and the pulse watched closely.

It must be remembered that the temperature will fall after removal from the bath, so that if a patient is placed in the water with a temperature of 107° F. he should be removed when it falls to 103° F. Twenty minutes is about the average time. It may be necessary to repeat the bath more than once.

The use of phenacetin, phenazone, and similar drugs is not advisable, as they are too depressing when associated with the cold-bath treatment.

If there is no convenience for giving a bath, a sheet wrung out in cold water may be wrapped round the patient, who is then rubbed with blocks of ice. This method also involves considerable shock and requires the same precautions in its employment.

**Vaccine- and serum-therapy.**—The question naturally arises why, if acute rheumatism is an infection due to a diplococcus of the streptococcal group, success has not hitherto followed the employment of vaccines and serums. It must be remembered that the acceptance of the microbic view of the cause of acute rheumatism has been very half-hearted in this country; although nearly twenty years have elapsed since the first papers on the subject were published, no serious attempt has yet been made either to corroborate or better the explanation. Had this been the case and had there been corroboration, doubtless by this time vaccines would have been used and many attempts made to procure a successful serum. I have treated obstinate cases with a vaccine, but can report no real success. I have also tried a serum in a considerable number of cases, but with no definite results. The whole subject, however, requires prolonged investigation, and at present is engaging close attention at the Hospital for Sick Children, Great Ormond Street.

Much has been written about the rheumatic phylacogen of Schäfer. Phylacogens are neither vaccines nor serums, but sterile aqueous solutions of metabolic derivatives generated by bacteria when grown in artificial media. I am not prepared to accept Schäfer's statement, which forms the basis of his phylacogen theory, that rheumatic infections are essentially mixed

## RHEUMATOID ARTHRITIS

infections. The phylacogen has been given subcutaneously and intravenously. The initial subcutaneous dose is 2 c.c., and this is gradually increased to 10 c.c. A dose is given daily unless there is some reaction. The intravenous dose is 0.5–5 c.c., repeated daily for six days. There may be severe reactions, particularly after intravenous administration, such as rigor, high fever, headache, malaise, and rapid action of the heart. I am very doubtful of the value of causing a severe reaction of this kind in a case of acute rheumatism, and personally prefer Nature to Science on such an occasion, and with such an alternative.

Radium in the treatment of acute rheumatism has not met with obvious success, although for the chronic forms much importance has been attached to its presence in spa-waters.

**Surgical treatment.**—The early surgical treatment of the joints in acute rheumatism has been advocated, with the end in view of preventing cardiac lesions arising by infection spreading from these local foci. The rationale of this seems to me faulty, and on this account I do not advocate the procedure. The cardiac infection more probably arises from the general invasion of the system than from the local lesion in the articulations. F. J. POYNTON.

**RHEUMATISM, CHRONIC.**—No useful purpose is served by keeping this title; it is mentioned here merely because it is commonly employed to denote any pain in joints, bones, muscles, tendons, or fasciae. It has no distinctive pathological basis, nor is it applicable to any symptom-complex or morbid entity. The recognition and separation of true rheumatism, of osteo-arthritis, of rheumatoid arthritis, of gout, and of inflammation of fibrous tissues, has robbed it of any scientific significance. Muscular pains are considered under MYALGIA.

FREDERICK LANGMEAD.

**RHEUMATISM, GONORRHOEAL** (*see GONORRHOEA*).

**RHEUMATISM, MUSCULAR** (*see MYALGIA*).

**RHEUMATOID ARTHRITIS.**—A subacute or chronic joint-disease of uncertain etiology, most common in young females, polyarticular and symmetrical in distribution, and involving small joints rather than large, and soft parts rather than hard, though the bone and cartilage undergo atrophic changes; it is associated with constitutional disturbance, runs a



## RHEUMATOID ARTHRITIS

prolonged and progressive course, and gives rise to gross deformities and crippling. Osteo-arthritis is described separately under its own name.

**Etiology.**—Many of the clinical features of the disease suggest an infective origin, but of this there is as yet no accepted bacteriological proof, though during the last twenty-five years numerous investigators have claimed the discovery of responsible micro-organisms. Many observers, including the writer, have obtained uniformly negative results on bacteriological examination of synovial fluid from affected joints, though trying all the usual laboratory media aerobically and anaerobically. In one case the writer cultivated a streptococcus from synovial membrane excised *intra vitam*, but numerous other cultures from similar material were sterile. The most promising recent work is that of Rosenow, who investigated the bacteriology of excised lymphatic glands in the vicinity of affected joints, and obtained positive results in a high proportion of cases, finding in most instances streptococci, but sometimes *B. welchii*, staphylococci, or *B. mucosus*. In some cases several different organisms were cultivated from the same gland. The very diversity of the organisms obtained, and their nature, arouse some scepticism as to their being concerned in the etiology of what many observers believe to be a specific disease.

It is doubtful if such chronic infective foci as septic gums, tonsils, ears, etc., are more common in sufferers from rheumatoid arthritis than in the rest of the community, and it is quite certain that there are many cases of this disease in which, however carefully we search, no sort of septic focus can be demonstrated. Pyorrhea or other septic foci are undoubtedly the cause of some cases of polyarthritis which can be arrested by wholesale dental extractions, or by vaccine or other treatment directed against the septic focus; but certainly some of these cases should be classed as "chronic septic arthritis" rather than as true rheumatoid arthritis. Chronic septic absorption from the colon has been held responsible for this, as for many other diseases, often on quite inadequate grounds.

In a certain number of cases the onset has followed more or less closely childbirth or a miscarriage, and sometimes it is associated with menorrhagia or other uterine disorders. Not infrequently some acute infection, such as influenza, seems to start the trouble. Worry or exposure to damp is blamed by some patients; but in many instances there is no

known exciting cause for the disease, which arrives "out of the blue."

Poncet and his school have tried to prove that rheumatoid arthritis is a sort of "para-tuberculosis," postulating a very attenuated tuberculous virus and a high degree of resistance on the part of the host, with resultant aspecific morbid changes. The evidence in support of this hypothesis is very flimsy, though there is no doubt that there is such a thing as a genuine chronic tuberculous polyarthritis, which may be mistaken for rheumatoid arthritis.

Malloch investigated the purin metabolism in rheumatoid arthritis, and in certain cases found disturbances akin to those of gout.

**Age.**—The disease may begin at any age; usually the onset is between the ages of 20–40. It is probable that "Still's disease" is rheumatoid arthritis of childhood.

**Sex.**—About 80 per cent. of the cases are females.

**Heredity.**—Owing to the confusion of nomenclature, most statistics as to rheumatoid arthritis are quite unreliable, so that we have no figures to guide us as to the influence of heredity in this condition. Some of the recorded instances of the occurrence of several cases of the disease in one family are hardly explicable on a basis of coincidence.

**Pathology.**—Two distinct types of the disease can be recognized—the *exudative* and the *atrophic*, of which the latter is usually, but not always, an end-stage of the former.

In the **exudative type** there is swelling not only of the synovial membrane and capsule of the joint, but also of the periarticular tissues, producing a characteristic spindle-shaped appearance of the joint. There is great proliferation of the synovial membrane, which is convoluted into numerous folds and fringes, and is vividly injected; microscopically the tissues are unduly vascular and diffusely infiltrated with round cells, but the most characteristic feature is a pronounced thickening of the arterioles and, to a less extent, of the venules. The perivascular sheath is chiefly involved in this thickening, but the lumen of the vessel is also encroached upon. A necessary consequence of the vascular occlusion is, eventually, a defective blood supply to the joint tissues, so that atrophic changes follow: the nutrition of the cartilage is impaired, and this tissue undergoes retrogressive changes, becomes gradually thinner, and ultimately may disappear entirely. Small areas of eroded cartilage may be

## RHEUMATOID ARTHRITIS

seen, but the eburnation, lipping, and osteophytic outgrowths characteristic of osteo-arthritis are not found; in fact, the changes are entirely those of destruction and absorption, without any attempts at repair as in osteo-arthritis. *Pari passu* with the absorption of cartilage proceed atrophy and sclerosis of the synovial membrane, the folds of which become adherent to one another and to the articular surfaces, giving rise in time to fibrous bands that cross the joint-cavity, which they may eventually obliterate. These ligamentous bands may become ossified, so that either fibrous or bony ankylosis may be formed. The atrophic process involves also the bone subjacent to the articular cartilage, and may extend for a considerable distance away from the epiphysis, the shell of compact bone becoming progressively thinner, and the meshes of the cancellous bone wider. This rarefaction of bone is always a prominent feature of advanced cases. The bones then become very brittle and are easily fractured. True osteophytes, such as are found in osteo-arthritis, are never present in rheumatoid arthritis, but in the latter disease, especially in its terminal stages, sharp spicules of new bone are apt to appear on exposed bony surfaces, e.g. the olecranon process, the patella, the coccyx, or the phalanges of the fingers. Wasting of the muscles supplying affected joints is extreme, and contractures appear early. Subluxations of joints are quite common. The synovial fluid is in excess during the earlier and more acute stages of the disease, when it is often possible to aspirate an ounce or more from the knee-joint. The fluid so obtained may be clear, but more often is slightly turbid, sometimes sufficiently so to give rise to a definite semi-purulent deposit on standing. In turbid samples of fluid the polymorphonuclear is the predominant cell, whereas in the clear samples lymphocytes, endothelial and polymorphonuclear cells are present in varying proportions, polymorphonuclear cells being usually in the majority.

The **atrophic type** has already been described as an end-result of the exudative type. It only remains to say that some cases are atrophic from the first, without any preliminary stage of swelling or exudation.

**Symptomatology.**—The onset is either insidious or acute. It may begin so very gradually that the patients have difficulty in dating it, and many years may elapse before they are seriously crippled. But not infrequently the

onset is acute and associated with fever to 102° or 103° F., and with swelling of many joints, so that for some time the disease is taken to be acute rheumatism, and not until it has persisted for many weeks without responding to salicylates is the true nature of the condition recognized. In the majority of cases the onset may be described as sub-acute: numerous joints are implicated, but there is at first little swelling of them, and fever will not be discovered unless the temperature is taken, when an evening rise to 99° or 100° F. will be found. One of the characteristic features of the disease is the tendency to exacerbations and remissions, which may alternate throughout a period of twenty to thirty years. The first joints affected are nearly always the proximal interphalangeal joints of the fingers, and most of the other joints of the hands and feet, including the wrists and ankles, are usually involved at an early stage. The knees and elbows tend to be involved next in order, and finally the hips and shoulders, though in an acute case most of the joints of the body may be implicated almost simultaneously. There is no tendency for the disease to fly from joint to joint, as in acute rheumatism, and once a joint has become seriously affected, it is doubtful if complete restoration of function, such as so often occurs in acute rheumatism, is possible. There is always a more or less obvious symmetry in the distribution of the joints involved. The vertebral joints, especially those of the cervical region, seldom escape, and the temporo-mandibular joints are also specially liable to be painful and stiff from time to time, as are the sterno-clavicular joints.

In the **exudative type** there is a pronounced swelling of the joint itself, and to a less degree of the periarticular tissues; this, combined with an early and well-marked wasting of the muscles above and below the joint, brings about a very characteristic spindle-shaped appearance, best seen in the case of the fingers, but often quite noticeable in the knees and elbow. The skin over the joint is not flushed, nor is it hot, and œdema of the periarticular tissues is never present, though in advanced cases there is often a little œdema of the extremities, especially of the feet; this is not an inflammatory œdema, but is rather a "trophœdema," and is associated with defective peripheral circulation. The joint-swells have a peculiar elastic feeling on palpation, so that it is often difficult to be certain, without aspirating, whether there is an excess of synovial fluid or

## RHEUMATOID ARTHRITIS

not. Even during the acute stages the joints are not very tender to palpation, differing in this respect from the joints of acute gout and acute rheumatism. Attempts at movement, active or passive, are extremely painful, and the range of movement is limited; this is due at a very early stage to involuntary muscular spasm, at later stages to muscular contractures, and eventually to fibrous or even osseous ankylosis. A very fine crepitus, quite unlike the coarse "scrunch" of osteo-arthritis, may be obtained during the early stages; in advanced cases a coarse crepitus may be present, but more often it cannot be elicited, owing to partial or complete ankylosis. Contractures and deformities are first manifest in the hands; various combinations of flexion and hyper-extension of the interphalangeal, metacarpophalangeal and carpal joints may occur, and ulnar deviation of the fingers is quite characteristic of, though not peculiar to, the disease. The wrists become fixed very early, and the knees tend to become flexed, a deformity which the patient invariably encourages by lying with a pillow under the knees. As the flexion of the knee becomes more pronounced, the tibia tends to undergo backward displacement, and often external rotation. The elbow-joints also become permanently flexed, and movements of the cervical spine are limited. Attacks of clonic spasm of the muscles of the lower limb are not infrequent, and are especially prone to occur during sleep, the pain induced thereby awaking the patient suddenly.

*Pain* is a very variable symptom, but it may be said that, as a rule, it is felt chiefly on movements, active or passive, and that so long as the joints are at rest it is not very severe. Some of the worst cases are attended by astonishingly little spontaneous pain. Changes of the weather can usually be foretold with some accuracy by rheumatoid patients, but on the whole they complain more of "stiffness" than of pain. Trophic changes of the skin and nails are invariable, and usually appear early. The skin of the hands and feet is thin and "glossy," and the palms and soles perspire freely; the extremities are usually cold and often slightly cyanotic. The nails are ribbed and very brittle. A patchy or diffuse pigmentation of the skin is sometimes observed. After the acute stage, the joint-swollings gradually disappear, and at the same time muscular wastings and contractions become more and more pronounced.

In the **atrophic type** of rheumatoid arthritis

(the *arthrite sèche* of French writers) the onset is very insidious, attended by paræsthesiæ and joint-stiffness, but there is little pain. Trophic changes in skin and nails are early and pronounced, as also are contractures and deformities. At no time are there any conspicuous joint-swollings.

**Constitutional symptoms.**—Fever has already been referred to. Persistent tachycardia, not necessarily associated with pyrexia, is sometimes observed. A certain degree of anæmia is always present during an acute phase, and not infrequently the patients are quite cachectic. Dyspepsia is not uncommon, and is sometimes due to persistence in some entirely unnecessary and unreasonable dietetic restrictions. Endocarditis and pericarditis have been described in this disease, but it is doubtful whether their appearance, or that of any other visceral manifestations, can be regarded as more than fortuitous. The spleen and lymphatic glands are sometimes enlarged slightly, but, in the writer's experience, this does not occur very often.

**X-ray appearances.**—The characteristic changes are diminution of the "joint-slits" (due to absorption of articular cartilage) and increased transparency of the bones (due to rarefaction of the latter). (PLATE 42, Fig. 5, facing p. 547.) Lipping and osteophytic out-growths are not seen.

**Diagnosis.**—During an acute phase the diagnosis can often be made at a glance, but difficulties arise even in this stage, and to a greater extent during the later quiescent stages.

*Acute rheumatism* may be closely imitated by early rheumatoid arthritis, and conversely an obstinate form of subacute rheumatism may simulate rheumatoid arthritis. Important differential points are the tendency of acute rheumatism to fly from joint to joint and to respond favourably to treatment by salicylates. Involvement of the temporo-mandibular, sternoclavicular and vertebral joints would point to rheumatoid arthritis, and evidence of endocarditis, pleurisy, or pericarditis to acute rheumatism.

*Osteo-arthritis*, when monarticular or oligarticular, is easily distinguished from rheumatoid arthritis, but the somewhat rare cases of polyarticular osteo-arthritis are more difficult to differentiate. The chronicity of the condition, with absence of fever or other constitutional disturbance, and, above all, the characteristic X-ray picture, with its osteophytes and its normal "joint-slits," may assist the diagnosis.

## RHEUMATOID ARTHRITIS

**Chronic septic arthritis** (including gonococcal arthritis).—During the acute stage the joints may be flushed and warm, and some degree of cedema at or near the joint is almost invariably to be detected. There is not the same tendency to symmetry, and when the initial acute stage is over there is an absence of the steady progression, with exacerbations and remissions, so characteristic of rheumatoid arthritis. Some obvious source of infection, e.g. pyorrhœa, gleet, or a pneumonic lung, is usually present.

**Chronic polyarticular gout.**—Here the diagnosis rests on a previous history of typical acute gout, and on the presence of definite tophaceous deposits.

**Still's disease**, as suggested above, is probably rheumatoid arthritis of childhood, and differs chiefly from that disease as it affects the adult in the presence of morbid glandular, and sometimes splenic, enlargement.

**Prognosis.**—The disease itself seldom if ever kills, and, when acute symptoms have subsided, is compatible with a very fair state of general health. Apart from this, the outlook is always exceedingly grave. Even when it is evident that the initial acute phase is over, with abatement of constitutional symptoms and diminution, or disappearance, of joint-swellings, there is always the possibility, or rather the probability, of a relapse. Nor is the severity of the symptoms at the onset any guide, for some of the most intractable and crippling forms of the disease begin in the most insidious fashion.

**Treatment.**—During the acute phases of the disease, and to a less extent throughout its course, *rest* of the affected joints is much the most important item of treatment, and unfortunately is one which it is most difficult to persuade the patient to carry out, obsessed as he invariably is with the idea that he must at all costs keep the joints moving to prevent their becoming stiff. If medical men would devote their attention to keeping joints with active disease *at rest in a good position*, instead of plying their patients with vaccines made in turn from the gums, urine, fæces and vagina, we should see less of the unfortunate crippling and deformities that are so common in this disease. It should be impressed on patients that it is better to have a quiet joint partially or completely ankylosed in a good position than one with active disease but more freely mobile. If necessary, splints must be used in order to obtain adequate rest for particular joints. Care must be taken to prevent flexion

and rotation at the knee-joint. Throughout the acute phases of the disease the patient must be kept strictly confined to bed, if necessary for many weeks.

The nutrition of the muscles must be maintained by gentle *massage*. It is rational to attempt to improve the circulation in the affected joints by hot-air baths, local or general, and these certainly are often beneficial, especially after the more acute symptoms have passed off. But this treatment must be administered with caution: it is a mistake to subject an already debilitated patient to an exhausting daily roasting. Another means of flushing the joints with blood is the congestive treatment of Bier; an elastic bandage is applied on the proximal side of the affected joints sufficiently tightly to impede the venous but not the arterial stream, and is removed after cyanosis, or even œdema, of the distal tissues is observed. The writer has not been favourably impressed by the results of this treatment.

**Diet.**—There is no evidence that dietetic restrictions are of benefit in this disease, except in so far as they may be necessary for gastric or intestinal disturbances. The diet should be planned to give adequate nourishment without producing obesity, and to obviate any tendency to either flatulence or constipation. The patients are usually better without alcohol.

**Climate.**—An equable, dry climate suits patients best, but if well clad they can tolerate a considerable degree of cold. Even during the acute stage, fresh-air treatment, day and night, is often very beneficial.

**Drugs.**—Guaiacol carbonate is advocated by several distinguished authorities. It is given in cachets or capsules in doses of 5 gr., gradually increased to 20 gr., three times a day. Aspirin is invaluable for its anodyne effects, though it has no influence on the course of the disease. Locally, an ointment of methyl salicylate 1 dr., menthol 10 gr. to 1 oz. of lanolin, is useful. Counter-irritation with Scott's dressing or by blistering is indicated in cases of persistent fluid effusion.

Constipation must be treated *secundum artem*. Some of these patients are obstinately constipated, and the satisfactory results sometimes obtained by the prolonged administration of laxatives, or of graduated enemata, lend support to the view held by some that the disease is due to alimentary toxæmia. Intestinal antiseptics, particularly kerol, are always well worth a trial. Colectomy has been advocated by Lane and his followers, but most

## RHINITIS

medical men will hesitate to adopt such drastic treatment without more convincing evidence of its efficacy.

In our ignorance of the etiology of the disease it is rational to investigate the whole body for septic foci, and to treat any that may be discovered. Pyorrhœa, septic tonsils, chronic catarrh of the nose and its sinuses, otorrhœa, leucorrhœa and urethritis or cystitis are the pathological conditions to be looked for. But in many cases none of these conditions is found, and when they do exist their treatment is often without influence on the course of a genuine case of rheumatoid arthritis. Much has been claimed for *vaccines* in this disease; there is no doubt that vaccine treatment in chronic septic arthritis may yield most gratifying results, but in rheumatoid arthritis the results are usually disappointing.

It is in chronic and advanced cases that *spa treatment* is most likely to be beneficial. The much-lauded mineral waters have probably no specific influence on the disease, though their aperient or diuretic properties may sometimes be beneficial. But skilfully administered baths of various kinds, followed by efficient massage, are helpful in reducing the deformities and increasing the mobility of joints which are not acutely inflamed.

*Orthopedic treatment* is not sufficiently employed in the later stages of this disease. Contractures of the knees can often be reduced by weight-extension, and, if this fails, by manipulation under anæsthesia. Care must be taken not to attempt any forcible reduction while there is any sign of activity in the arthritis, and it must be remembered that the femur is so brittle that it may be fractured more readily than the articular adhesions. When the deformity has been reduced, the joint may be allowed to ankylose again, extension being kept up by a rigid splint; or, in carefully-selected cases, a hinged calliper splint may be applied. In either case, a bedridden patient may become ambulatory, and the results obtained are sometimes brilliant.

MAURICE CASSIDY.

**RHINITIS.** Of inflammation of the nasal mucous membrane seven distinct forms are recognized, beginning with—

### 1. ACUTE CATARRHAL RHINITIS

**Etiology.**—The commonest organisms producing this disease are the *Micrococcus catarrhalis*, the *pneumococcus*, and various types of *streptococci* and *staphylococci*.

**Symptoms.**—The first symptom complained of is usually a feeling of fullness in the nose accompanied by irritation. In a few hours the nostrils become obstructed, and a mucous or muco-purulent discharge makes its appearance. Headache and general malaise are also present, and the temperature is usually raised. Secondary results, such as deafness, pain in the ears, pharyngitis and laryngitis may supervene in either a slight or a very severe form.

**Diagnosis.**—On examination the mucous membrane is found to be acutely inflamed and swollen, the swelling of the turbinal bones being usually very considerable and perhaps blocking completely the interior nasal meatuses. Both nostrils are always affected.

**Treatment.**—The treatment which gives most relief is a spray of methyl-salicylate (5 per cent.) in paroline every four hours, combined with the internal administration of aspirin or quinine. A purgative should always be given at the onset, and repeated as often as necessary. In small children inhalations of eucalyptus and compound tincture of benzoin are most useful.

It must be remembered that acute rhinitis is often the first manifestation of exanthemata such as scarlet fever and measles; when it is detected in children a careful watch must therefore always be kept for the appearance of any characteristic rash.

### 2. CHRONIC RHINITIS

**Etiology.**—This disease, which is usually bilateral in character, occurs most commonly in patients suffering from hypertrophic conditions of the middle and inferior turbinal bones (*see* NASO-PHARYNGEAL OBSTRUCTION), septal deflections and spurs, or suppuration in one or more of the nasal sinuses. It is also met with when frequent attacks of acute rhinitis occur, and in patients suffering from adenoids. Excess both of alcohol and of smoking is sometimes responsible.

**Symptoms.**—The most common symptom is nasal obstruction, which varies in degree from slight unilateral obstruction to complete blocking of both nostrils. The amount of obstruction is not constant, and may vary during the day, being usually worse at night. There may be headache and loss of smell if the middle turbinal bone is sufficiently enlarged to press upon the septum and obliterate the olfactory cleft. A mucous or muco-purulent discharge, slight or profuse in quantity, is always present.

## RHINITIS

Secondary symptoms due to involvement of the ear, pharynx and larynx usually follow sooner or later.

**Diagnosis.**—On examination the septum and turbinal bones are seen to be covered by inflamed mucous membrane. If the disease is of some standing, all or any of the hypertrophic conditions described under NASOPHARYNGEAL OBSTRUCTION may be recognized.

**Treatment.**—When the disease is secondary to sinus suppuration, deflections of the septum, or hypertrophic rhinitis, the primary condition must first be corrected by appropriate treatment. In mild cases a douche of normal saline solution employed morning and evening usually gives quick relief. In those which do not react readily to this treatment, painting the mucous membrane daily with the following solution generally effects a cure :

R̄ Iodī gr. iv.  
Menthōl gr. viii.  
Paroleīne ad ʒi.

### 3. MEMBRANOUS RHINITIS (NON-DIPHTHERITIC)

**Etiology.**—This condition, as a rule, only affects patients who are suffering from acute fevers, such as measles and scarlet fever, and is due in most cases to streptococci, pneumococci, or staphylococci.

**Symptoms.**—A blood-stained purulent discharge is invariably present, and, owing to the presence of the membrane, obstruction is always complained of. Constitutional symptoms are well marked.

**Diagnosis.**—On examination a greyish membrane, which bleeds when attempts are made to remove it, is seen upon the septum and turbinal bones. It is distinguished from that of *nasal diphtheria* by the absence of the Klebs-Löffler bacillus.

**Treatment.**—A spray of peroxide of hydrogen, followed by irrigation with the following solution, should be employed :

R̄ Sod. bicarb. | āā gr. xxx.  
Sod. chlor. |  
Acid. carbol. gr. ii.  
Aq. ad ʒss.  
ʒss. to ʒxx. of warm water.

After the douche, an oily spray, such as menthol (5 per cent.) in paroleine, helps to prevent the membrane from reforming.

When, as sometimes happens, this disease shows a tendency to become chronic, an auto-genous vaccine is indicated, in addition to the local treatment.

The general treatment consists in the administration of tonics, purgation when necessary, and rest in bed.

### 4. PURULENT RHINITIS

**Etiology.**—Purulent rhinitis originates from a variety of causes. In children the commonest causative agents are foreign bodies and adenoids, whilst in adults sinus suppuration, or syphilitic or tuberculous lesions, are responsible for the majority of cases. Patients suffering from scarlet fever, measles and other acute infections often develop purulent rhinitis.

**Symptoms.**—A purulent discharge is always present; it may be unilateral when due to foreign bodies or suppuration in one or more of the accessory sinuses, or bilateral as when due to adenoids. There is more or less complete nasal obstruction when the disease is produced by adenoids or a foreign body. When sinus suppuration is the cause, any or all of its special symptoms may be associated.

**Diagnosis.**—A bacteriological examination of the discharge excludes *diphtheria*, whilst a careful examination of the nose and the demonstration of the absence of any membrane distinguish the malady from *membranous rhinitis*. The absence of *adenoids* and *foreign bodies* is easily proved by thorough anterior and posterior rhinoscopy. Should syphilis or tuberculosis be suspected, a Wassermann reaction and a detailed examination of the patient for syphilitic or tuberculous lesions elsewhere assist in making a correct diagnosis.

The **prognosis** is good except in patients suffering from tuberculous disease, in which it depends upon the extent and severity of tuberculous lesions elsewhere. When tuberculous rhinitis is primary, energetic local treatment usually gives good results.

**Treatment.**—The treatment is obvious when foreign bodies or adenoids are present; in the latter case, however, if the patient's general condition is poor, it is wise to employ local treatment for a time and even to use an auto-genous vaccine before operation is resorted to, owing to the risk of septicæmia, a complication which is especially likely to occur in patients whose resisting powers are much below the normal.

In syphilitic and tuberculous cases the appropriate treatment must be resorted to.

Gentle irrigation with the following lotion should be carried out three times a day :—

## RHINITIS

R $\bar{y}$  Sod. bicarb. } 55 gr. xxx.  
Sod. chlor. }  
Acid. carbol. gr. ii.  
Aq. ad 3ss.  
3ss. to 3xxx. of warm water.

When the discharge is very profuse a few drops of peroxide of hydrogen should be instilled into each nostril before the douche is used.

Should the condition show any tendency to become chronic, the use of an autogenous vaccine is usually of definite assistance in the patient's recovery.

### 5. CASEOUS RHINITIS

This is a variety very rarely met with, and its exact origin is not known.

**Symptoms.**—The chief symptoms are obstruction, a bad odour emanating from the nose, and an offensive, nauseating breath. Headaches may occur and may be very severe, and there may be also a purulent discharge. Partial or complete loss of smell occurs when much destruction of the septum or of the mucous membrane covering it takes place in the olfactory area.

**Diagnosis.**—Examination of the nose reveals a caseous mass which is usually situated in the olfactory cleft. When it has been present for a considerable time it may have caused ulceration of the septum, which finally perforates, so that the mass is seen to be protruding into the opposite nostril.

**Treatment.**—The mass must be removed from the nostril under a general anæsthetic, and very careful after-treatment is necessary when there has been ulceration of the opposing surfaces of the septum and turbinal bones, so that the formation of adhesions may be prevented. When the patient cannot be kept under constant observation for a few weeks after operation, rubber splints should be inserted, as described in the article on NASOPHARYNGEAL OBSTRUCTION.

A douche of normal saline solution three times daily followed by a spray of menthol (5 per cent.) in paroline should be ordered and continued until complete healing has taken place.

The prognosis is always good.

### 6. RHINITIS SICCA

"Dry rhinitis" occurs in patients who are working in a hot, dry atmosphere, and is therefore frequently met with in stokers. When too much of the inferior turbinal bone has been

removed by operation, rhinitis sicca sometimes supervenes. It may also complicate debilitating diseases such as anæmia, or follow indulgence in alcohol. Deafness, middle-ear suppuration, pharyngitis, and laryngitis may be added in the later stages.

**Symptoms.**—Dryness of the nose is the chief symptom, and this is usually accompanied by irritation and discomfort due to the crusting which takes place in the naso-pharynx and pharynx as well as in the nose. Epistaxis occurs when crusting and subsequent ulceration of the septum have developed, the latter condition being caused by the constant attempts of the patient to dislodge the crusts. This ulceration may ultimately lead to perforation of the septum.

**Diagnosis.**—The mucous membrane is dry and has a glazed appearance, and the crusts, which are usually small in size, are seen on the septum and also on the anterior ends of the turbinal bones. Ulcers, or actual septal perforations, may be found, and are almost always situated on the anterior part of the septum. There is no possibility of confusing this disease with atrophic rhinitis, in which much atrophy of the turbinal bones and large foul-smelling crusts occur.

**Treatment.**—A douche of normal saline solution should be used morning and evening, and to prevent crust-formation the nose should be sprayed with a solution of methyl-salicylate (5 per cent.) in paroline after the douche has been used. Some cases react much more rapidly to treatment if the nose is painted daily with the following:—

R $\bar{y}$  Iod. gr. iv.  
Menthol gr. viii.  
Paroline ad 3i.

Once the disease is definitely established, a permanent cure is impossible, but the symptoms can be very much relieved by the above treatment.

### 7. ATROPHIC RHINITIS (OZÆNA)

**Etiology.**—This form of rhinitis usually affects patients suffering from longstanding sinus suppuration, or from syphilis or tuberculosis. Sometimes the disease seems to follow purulent rhinitis dating from scarlet fever or from measles in early life. The actual factor concerned in its production is, however, unknown.

**Symptoms.**—The most distressing symptoms are the formation of foul-smelling crusts, and a very characteristic foul odour which causes the patient to be shunned by his friends.

## RHINORRHOEA

There is usually much irritation in the nostrils, and bleeding may arise from ulceration of the mucous membrane. Coughing is usually very troublesome, and is due to the dryness of the pharynx and larynx, and to actual crust-formation in these two regions; a profuse muco-purulent and purulent discharge is always present. Aural, laryngeal, and tracheal complications follow, and in many cases the accessory sinuses of the nose become infected, the symptoms of suppuration in one or more of these sinuses being added to those already described. In old-standing cases actual deformities, such as falling in of the bridge of the nose and dilatation of the nostrils, supervene.

**Diagnosis.**—The diagnosis is not difficult, as the large offensive crusts and the very marked atrophy of the turbinal bones are sufficiently characteristic. A Wassermann reaction and examination of the patient for tuberculous lesions elsewhere are useful diagnostic procedures. When the atrophic rhinitis is unilateral it is always due to disease of one or more of the nasal accessory sinuses.

**Treatment.**—The nose should be irrigated with normal saline solution three times a day, and more often if necessary. When the crusts are not easy to dislodge, some peroxide of hydrogen should be sprayed into the nostrils a few minutes before the douche is used. An oily spray should be used after the syringing, since it tends to prevent crust-formation, the best being —

- 17 Menthol gr. viii.  
Ol. eucalypt. ℥iij.  
Ol. cinnam. ℥iij.  
Paroleine ad ʒi.

In some cases much permanent relief can be given to the patient by building up the atrophied turbinal bones by submucous injections of solid paraffin.

No form of treatment produces a permanent cure in well-established cases, but the symptoms can be much relieved by persistent and energetic measures.

G. N. BIGGS.

## RHINOPHYMA (see ACNE ROSACEA).

**RHINORRHOEA.**—A discharge of watery fluid from the nose, which may either be cerebro-spinal fluid or arise from the nasal mucous membrane.

1. **HYDORRHOEA.**—Nasal rhinorrhœa is met with only in patients of a very neurotic temperament. They complain of repeated attacks of a profuse mucinous discharge from the nose.

## RHINOSCLEROMA

It occurs during the day, and in some cases is accompanied by much local irritation and sneezing. The symptoms generally pass off at night. These cases are distinguished from those of cerebro-spinal rhinorrhœa by the presence of mucus in the discharge.

**Treatment.**—Any nasal abnormalities such as a deflected septum, enlargement of the inferior or middle turbinal bones, or areas of hyperæsthesia must be rectified by appropriate treatment, and calcium lactate should be given in large and rapidly increasing doses internally. When there are definite local lesions their correction is usually followed by considerable relief from the symptoms, and occasionally by a cure. In the absence of any local condition the outlook is not very promising, although some cases are relieved, or even cured, by the administration of calcium lactate.

2. **CEREBRO-SPINAL RHINORRHOEA.**—This condition occurs as a unilateral or bilateral watery discharge which is very profuse and persistent in character and comes on without warning. It is unaccompanied by local irritation, nor is nasal obstruction complained of unless the disease is complicated by some other nasal condition, such as hypertrophic rhinitis. The discharge is of low specific gravity, free of mucus, and reduces Fehling's solution.

**Treatment.**—Local treatment should never be undertaken, even when the condition is complicated by a deflected septum or enlargement of the turbinal bones, as the risk of infecting the meninges is very great. Tonics are indicated, but the disorder is practically incurable.

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**RHINOSCLEROMA.** A cheloid-like tumour consisting chiefly of small round cells embedded in a fibrillated stroma, and growing from the deeper layers of the mucous membrane of the nose or the skin near the anterior nares. It is moderately vascular. Its etiology is uncertain. Nasal obstruction is gradually produced by steady but slow extension of the original swelling, until perhaps the obstruction is complete. As the growth increases, external deformity and severe headaches always occur, and sometimes attacks of nasal hæmorrhage are complained of. Ultimately, as well as the nasal cavities, the lips, gums, palate, and pharynx may be infiltrated and become rigid and scar-like, while the glottis is narrowed. In the early stages a smooth and red swelling can be seen by anterior rhinoscopy; it may be found in any position in the nose, and is



## RICKETS

quite firm when touched with a probe. To establish the diagnosis a piece of the growth should always be removed for microscopical examination, to exclude other forms of growth and syphilitic or tuberculous granulomata, although in the latter diseases the presence of tuberculous disease in the lungs, or of syphilitic lesions elsewhere combined with a positive Wassermann reaction, will enable a fairly accurate diagnosis to be made.

**Treatment.**—If practicable, the growth should always be removed by operation; in cases in which complete excision cannot be effected, as much as possible of the growth should be removed in order to relieve symptoms. Arsenic should be given in rapidly increasing doses in inoperable cases, and whenever only partial excision is possible; it is said to have a beneficial effect. The same is claimed for radium and X-rays, so that these agents should always be given a trial. Local recurrence has invariably followed removal sooner or later, but generalization is unknown.

G. N. BIGGS.

**RHINOSCOPY** (*see* NOSE, EXAMINATION OF).

**RICH'S REACTION** (*see* ELECTRICAL REACTIONS).

**RICKETS.**—A general disease of uncertain etiology, occurring in childhood, manifesting itself chiefly by abnormal development of the skeletal system, by muscular hypotonia, and by diminished resistance to infection, accompanied by constitutional disturbance, and prone to lead to deformities.

**Etiology.**—A complete explanation has still to be found, and no detailed exposition of the conflicting hypotheses can be given here. Among various divergent views, that which has received widest acceptance is that rickets is due to an error in diet and is, in fact, a *nutritional disorder*. Deficiency in fat or in protein and excess in carbohydrates have each in turn been regarded as the most important error. A good deal can be said for and against each of these views, and it cannot be doubted that rickets is more likely to occur in children who are artificially fed and are therefore more liable to be fed wrongly and to receive an ill-balanced diet. Patent foods, which, generally speaking, are low in fat content, if persisted in may lead to the development of rickets; and condensed milks, to which sugar has been added, and which consequently contain a considerable carbohydrate excess, are still more deleterious

in this connexion. It is held by some that the surplus carbohydrates cause rickets through the intermediation of the toxic products which result from their fermentation or are produced by organisms which thereby find a suitable medium. Again, too protracted feeding at the breast appears to be responsible for some examples of rickets in the temperate zone; on the other hand, in India and Japan infants are often fed entirely at the breast for two years or longer, but escape the malady. The work of E. Mellanby explains most of the difficulties which surround the subject. By feeding experiments on puppies he has come to the conclusion that the defect in diet is not essentially a relative lack or excess of fat, protein, or carbohydrate, but consists in a relative insufficiency of an accessory food factor (vitamin) which accompanies fat in its distribution; in other words, that rickets must be added to the list of "food-deficiency diseases." Fat-soluble A is the accessory food factor which has this distribution, but the work which has hitherto been done on this substance connects it with growth, whereas, according to Mellanby, those animals which grew quickest were the most prone to rickets.

According to another hypothesis, rickets is due not to errors in diet but to faulty hygiene and confinement, with a consequent lack of exercise. Findlay's experiments appear to show that, with animals fed on a particular diet, only those which are kept in confinement develop the disease, while the data of systematic investigations by Noel Paton, Findlay, and Miss Robertson certainly indicate that insufficient air-space and want of exercise are at least important factors. One other hypothesis only will be mentioned: it supposes that rickets is due to infection, but at present it is based on inadequate evidence.

Though breast-fed infants are not immune, probably because of poverty of the milk or too prolonged suckling, rickets most commonly appears in artificially-fed children between six and eighteen months of age, and early cases are most frequently met with in the spring months. Geographically, rickets is most prevalent in the temperate zones, i.e. in the industrial areas of the globe, but is by no means confined to them. It is common in the British Isles, and especially prevalent and severe in Glasgow. Both faulty hygiene and bad feeding commonly precede it, and the poorer population is chiefly affected. A familial tendency is probable, for of two families, equally satis-

## RICKETS

factorily fed and housed, and with similar habits as to hygiene, every member of one may develop the disease in a severe form, while the other may remain free.

**Symptomatology.**—In the early stage no symptoms referable to the bones are present. The infant is pale, fretful, whining, and restless, and resents examination and handling. Sweating is profuse, and from the head may be so copious as to soak into the pillow. The weight of the bedclothes apparently causes discomfort, and they are repeatedly kicked off. Although weight may be maintained or continue to increase satisfactorily, the muscles are soft and flabby, a hypotonia which, with the associated listlessness, diminishes the infant's activity and indirectly assists in impairing appetite and development. The blood shows the changes of a secondary anæmia, which may be severe. The disease is often unrecognized until the characteristic changes in bones become manifest.

**Osseous system.**—The peculiar bony changes are caused by excessive though partially abortive activity of the osteogenetic areas and by softening. Thus arise enlargement of epiphyses, bending of bones which are subjected to mechanical forces, and fracture. The fractures are usually of the "greenstick" variety, but may be complete, and are generally united by extensive formation of callus. The epiphyseal enlargement, likened by Trousseau to the nodes produced by cutting circularly round the bark of a tree, may be very general or may only be noticeable in a few bones, particularly the lower ends of the radii, tibiae, and femora, and the costo-chondral junctions. Bending and moulding take place both in the long bones and those which form the thoracic cage and the pelvis. The long bones which suffer most are those of the legs, which bear the weight of the body. The *femora* are commonly bowed outwards and forwards if the child has stood or walked, and may bend directly forwards if its habit has been to sit on its mother's knees with its legs unsupported. A cross-legged "sartorial" position is often assumed by rickety infants, so that the femora become rotated outwards and the feet inverted. The *tibiae* and *fibulae* are either curved outwards or forwards, or in both these directions, just above the ankle; the bending of the tibiae forwards and outwards renders their crests prominent and produces the so-called "ploughshare" appearance. At the *knees genu varum* or *genu valgum* or *genu recurvatum* may be

caused; while at the *hip coxa vara* and, less commonly, *coxa valga* may result. In the former the angulation of the neck to the shaft is less than  $120^{\circ}$ , and in *coxa valga* is greater than  $140^{\circ}$ . The bones of the upper limb are usually less affected by bending, but in a child who crawls about may yield to pressure. The *clavicles* are often bowed forwards, and more frequently show fracture than any other bone; it is almost always of the "greenstick" variety. The *humeri*, *radii*, and *ulnae* bend forwards and outwards, while the radius has often a spiral twist, causing continual pronation.

**The skull.**—Prominent bosses of bone at the ossifying centres are met with. Usually the chief bosses are frontal and parietal, which, with the intervening valleys caused by the sagittal and coronal sutures, present the characteristic "natiform" or "hot-cross bun" skull. The forehead often projects. Fontanelles and sutures are late in closing, especially the former, and in their neighbourhood dimpling of the bone (craniotabes) may be elicited by pressure. The other variety of craniotabes, in which lacunae appear in the centre of ossified bone, away from the foraminal or sutural margins, is less definitely connected with rickets. The face is small in relation to the cranium. The *upper jaw* is often narrow and pointed anteriorly, while the *lower jaw* is squarer than usual, imperfect apposition of the teeth of the two jaws being thus brought about. The *teeth* themselves erupt late and are badly formed, often showing pitting and discoloration, and readily becoming carious.

The *chest* may be greatly distorted. Anteriorly at the costo-chondral junctions is the rickety "rosary" due to their enlargement. The beads are most marked at the junction of the fifth and sixth ribs with their cartilages. Some thickening of the ribs at their junctions is to be expected, but in rickets the prominences are often considerable and their sides steep. Just behind the line of union between cartilage and bone the ribs may yield to atmospheric pressure, and thus is produced a scooped-out hollow on each side, running from the second or third rib above, downwards and outwards to the hypochondria. In front of this groove the sternum and costal cartilages appear to be projected forwards, and, due to its presence, a transverse section of chest is fiddle-shaped, or resembles a vertical section of a cottage loaf, the base of which is posterior. A pigeon chest is not caused by rickets alone, but is likely to develop in a rickety child who

## RICKETS

has also naso-pharyngeal obstruction. Another groove, the rickety zone or Harrison's sulcus, encircles the anterior part of the chest above its outlet at the level of the upper margins of the abdominal viscera, and is also a result of atmospheric pressure. The lower costal margin is often everted, sloping out from a contracted chest to a prominent abdomen. Greenstick fractures at the rib angles occur in severe cases and explain the "posterior beads." Over the præcordium the thorax may bulge somewhat, but even so the heart may be displaced and, post mortem, may show a so-called "milk spot" due to pressure by a bead.

The *pelvis* in children who have walked may be narrowed antero-posteriorly, while its shape may be altered by pushing inwards of the acetabula, which are also generally nearer the front than normally. In this way varieties of flat pelvis and of trifoliate pelvis arise and may seriously interfere with parturition in later life. The iliac crests are thickened and may bend outwards, and the pubic arch is widened.

The *spine* may be curved, but escapes in mild cases. The most characteristic bend is kyphotic; it may be extreme, the "cut back," and extend from the lower cervical vertebræ to the sacrum. Its steepness may suggest spinal caries, but, as a rule, it disappears if the child is held up. Lateral curvature is common, but lordosis is less so, and probably compensatory to other deformities.

The bones of the *hands* and *feet* show little change, but spindle-shaped swellings may be seen in the fingers between the joints, which give the digits a curious beaded appearance. X-rays show that these swellings are entirely made up of soft structures, and that the bone takes no part in them.

For the *radiographic appearances* of the bones, see X-RAYS, DIAGNOSTIC USES OF, and PLATE 42, Fig. 6, facing p. 547.

**Muscles and tendons and ligaments.**—Muscular hypotonia is evident to palpation and, in association with laxity of tendons and ligaments, no doubt plays a part in the production of such deformities as the spinal curvatures, genu varum, and flat-foot. The same factors permit of undue mobility of the joints, a feature so obvious in some cases that they have received the name of "acrobatic rickets." Thus, it is not unusual for a rickety infant to sit for long periods with its toes in front of its face or in its mouth, while the feet may often be made to appose behind the head without difficulty. Some children can be folded up

like a pocket camera. The "double-jointedness" of the fingers may allow the digits to be extended on the metacarpals to beyond a right angle. Want of tone of the muscles of the abdominal wall explains in part also the prominent abdomen or "pot-belly" of rickets, but to this the contracted chest with displacement of viscera downwards and the hypotonia of stomach and intestine also contribute. The recti abdominis are often parted in the midline and permit of a linear ovoid protrusion between them, or of a more definite supra-umbilical hernia.

**Abdomen.**—The prominence and the changes in the abdominal wall have already been mentioned. A hyper-resonant note is often obtained, due to distended bowel, and the abdomen is particularly soft and yielding to palpation. The stomach and the small and large intestine may all be dilated, sometimes to a considerable degree. The liver and spleen may both be palpable; it is doubtful whether the view that these organs are often enlarged has not arisen through their readier accessibility. To the unaided eye the stools appear normal, but chemical examination has shown that there is excess of the alkaline earths, especially of calcium. Constipation is common, and is probably explained by the weakness of the abdominal wall and the intestinal muscle.

**Nervous system.**—The child dislikes being handled, and in active cases there appears to be some tenderness of the bones, which may explain the distaste for the bedclothes. Convulsions are often seen in rickety children, and would appear to be more easily produced in them by trivial causes, though rickets alone is not a sufficient explanation. The group of phenomena now called spasimophilia—tetany, laryngismus stridulus, and facial irritability—were at one time regarded as symptoms of rickets and are generally found in this country in children with this disease. Mental backwardness is a customary accompaniment of the tardy physical development, but the children often appear precocious in manner for their age.

An important feature of rickets is **lowered resistance to infection**. Catarrh of the nose and naso-pharynx, with the supervention of adenoids and tonsillar overgrowth, is particularly common and often leads to enlargement of lymphatic glands in the neck. Bronchitis is a frequent complication and is apt to be protracted and recurrent; the deformity of the chest renders it particularly severe and

## RICKETS

liable to be accompanied by considerable pulmonary collapse. Broncho-pneumonia is a serious complication, and particularly fatal in rickety infants. Dyspeptic disorders and gastro-enteritis readily occur and are combated with difficulty.

**Pathology.**—Examination of the bones reveals great preparation for the formation of bone but unequal achievement. The spongy tissue is increased and the irregular ossification is accompanied by undue vascularity, while soft vascular areas are also visible in the cartilage. The diaphyseal-epiphyseal junction is broadened and irregular.

**Microscopically**, the zone of multiplying cartilage cells is seen to be deeper than normal, the cells being arranged in rows which are much more irregular, while they vary greatly in size, some being abnormally large, others small. Calcification, too, is very irregular both in matrix and cartilage cells, so that uncalcified and calcified foci are intermingled instead of presenting a continuous frontier. The cartilage becomes vascularized. Beneath the periosteum is also a vascular cellular layer of abnormal thickness, and the new tissue which is formed, though considerable, is soft and vascular and unlike true bone, from the irregular and abortive deposition of calcium salts; it is commonly called "osteoid tissue." This formation of osteoid tissue explains the thickening which occurs in the ossifying centres of the flat bones, such as those of the cranium.

The **diagnosis** of developed rickets is not difficult, but the disease is often overlooked in its early stage before bony changes occur. Sweating, pallor, querulousness, loss of sleep, and flabbiness of muscle should always suggest the possibility of early rickets in an infant of about six months, and should lead to suitable treatment. The diagnosis may be confirmed by X-rays. *Achondroplasia* differs especially in being a congenital disease, in the relative shortening of the upper arms and thighs as compared with the trunk and more distal parts of the limbs, in the absence of constitutional symptoms, and in the X-ray appearances (see X-RAYS, DIAGNOSTIC USES OF). *Osteogenesis imperfecta* is often associated with fractures and consequent deformity, but in an unmixed case the characteristic rickety changes are absent. The prominent abdomen and general distension of bowel may suggest *celiac disease*, especially as the latter may be accompanied by rickets and dwarfing, but the large, grey, soapy stools of *celiac disease*

are characteristic. The rickety head is often mistaken for that of *hydrocephalus* because of its size, but in the latter there is a top-heavy appearance, the fontanelle bulges, sutures are more patent, and the rickety bosses are absent.

**Prognosis.**—Rickets is fatal only through secondary infections or disorders such as broncho-pneumonia, gastro-intestinal disorders, laryngismus stridulus, or convulsions. Unless detected and treated early it is apt to be protracted, and may continue until the end of the first dentition. Disappearance of the constitutional symptoms and increasing firmness of muscles mark the end of its activity. The enlarged epiphyses and deformities, even when considerable, tend slowly to disappear as age advances, and in the majority of cases have gone by puberty. Spinal curvature, genu valgum and varum, flat-foot, contracted chest and pelvis, and other gross deformities may, however, persist throughout life, as may also an abnormal laxity of joints. The degree of residual deformity is an index of the care given to the treatment.

**Treatment.**—*Fresh air and proper feeding* are important. Should rickets develop in a breast-fed child it is an indication for additional food and the beginning of weaning. In an artificially-fed infant particular care should be taken to see that the fat and protein content is satisfactory and that the carbohydrate is not in excess. Patent foods and condensed milk are not permissible. Animal fats are particularly indicated, and of these cod-liver oil is the best both on experimental and on clinical grounds. Cream or butter may be added in the bottle, but cod-liver oil is more pleasantly given afterwards in a spoon. Milk should be continued for the whole of the rachitic period, but not in excess of 1½ pints daily. After the ninth month the diet may be extended, and rusk and milk, the yolk of an egg, and meat gravies added. After twelve months, rusk and butter, bread and butter, stewed apple and prunes, and milk puddings are permissible, with the yolk of an egg at least once daily.

**Clothing.**—The clothing should be loose and light so that respiratory and abdominal movements are unrestricted. At night pyjamas and socks are necessary because of the tendency for the bedclothes to be kicked off.

**Exercise and massage** are valuable curative measures, but should always be followed by sufficient rest. The adjustment between rest

## RICKETS

and exercise is special to each child. In a mild case complete rest is neither possible nor useful, but in more severe cases, with bending of the bones as a result of sitting or walking, or of wrong postures while lying down, splinting by long external splints is called for; the splints should be worn both day and night, for the child often assumes faulty positions in bed, and delay in adjusting the splints in the morning may do much to counteract the good effect. Splints should be taken off morning and evening to permit the limbs to be bathed in salt water (rock salt 1 oz., water 2 gallons) and well rubbed with a towel or massaged. In most cases splinting of the legs alone is necessary.

**Drugs.**—Cod-liver oil is of most value. It may be given alone or as an emulsion with maltine or hypophosphites, e.g. :—

℥v Ol. morrh. ℥xxx.  
Sod. hypophosph. gr. ss.  
Calc. hypophosph. gr. ss.  
Ol. cassiæ ℥i.  
Glycer. ℥vi.  
Trag. q.s.  
Aq. dest. ad ℥i.  
T.d.s.

Iron may be added for anæmia in the form of syr. ferr. phosph. co. (15 min.). Several excellent emulsions of cod-liver oil, with and without malt or iron, are on the market. Sodium bromide (1–3 gr.) is valuable as a temporary measure if there is restlessness or insomnia.

**Operative procedures** for the correction of deformities should be postponed until after the seventh year, but much may be done by *orthopædic measures* in such cases to counteract the effects of abnormal lines of force.

### ADOLESCENT OR LATE RICKETS

This is rare. It begins usually after 6 years of age, and generally before 12. It is seldom primary, and more often due to relapse or recrudescence of a former rickets which has persisted, though latent or nearly so. It is commoner in girls than in boys, and has been recorded as following celiac disease. In its main features it resembles ordinary rickets, but, since the ossification of the skull is complete, skull changes are absent.

FREDERICK LANGMEAD.

**RICKETS, FŒTAL** (see ACHONDROPLASIA).

**RIGA'S DISEASE** (see STOMATITIS AND GLOSSITIS, p. 279).

## RINGWORM

**RIGOR MORTIS** (see POST-MORTEM EXAMINATIONS IN MEDICO-LEGAL CASES).

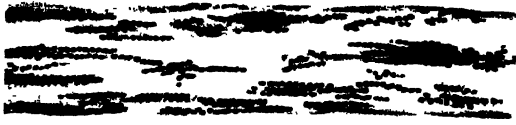
**RINGWORM.**—A disease of the skin, hair, or nails (PLATE 28, Figs. 1, 2, 3, 5, and PLATE 29), caused by a vegetable fungus.

**Etiology.**—Ringworm of the scalp is essentially a disease of children, and tends to disappear about the age of 15. It is common in schools and other institutions, through which it spreads by direct contact or by means of caps, hair-brushes, pillow-cases, etc. In adults, ringworm of the body and hair of the beard is fairly common, and is generally of animal origin. The groins, axillæ, fingers, and toes may also be attacked, and in rare cases there is extension to the mucous membranes.

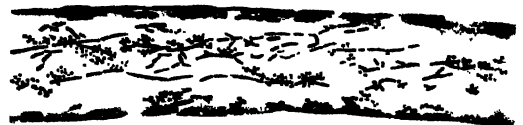
**Pathology.**—There are three large groups of fungi causing ringworm, the Epidermophytons, the Microsporons, and the Megalosporons or Trichophytons, the last being subdivided into *Tr. endothrix* and *Tr. ectothrix* or *endo-ectothrix*, according to the disposition of the fungus inside, or both inside and outside, the hair. The classification into small- and large-spored varieties is not strictly accurate on account of the great variation in size of the spores.

**Microsporosis.**—The most important member of the microsporons is *M. audouinii*, which is of human origin and causes at least 90 per cent. of the cases of ordinary scalp ringworm in this country. The chief animal microsporons communicable to man are derived from the dog (*M. canis* or *lanosum*), from the cat (*M. felineum*), and rarely from the horse (*M. equinum*). The microsporons are characterized by small round spores (2–4  $\mu$ ) scattered over the surface of the hair or closely packed together so as to form a sheath around it, and a wavy, irregularly-jointed branching mycelium terminating in a fringe above the hair-bulb. The fungus first attacks the epidermis, and invades the hair from above downwards, causing erosion of the cuticle and disintegration of the shaft. On Sabouraud's proof-agar microsporons give smooth white downy cultures.

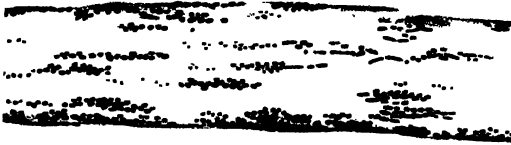
**Trichophytosis.**—The trichophytons are distinguished by short, regularly-jointed cubical mycelial elements (the term spore is a misnomer) arranged in chains. Usually these elements are larger than those of the microsporons, but there is a group (*Tr. microides*) in which they are small although they keep the characteristic chain-formation of the trichophytons. The



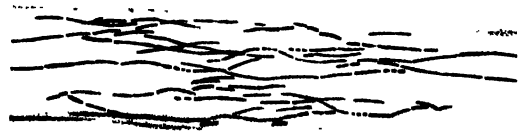
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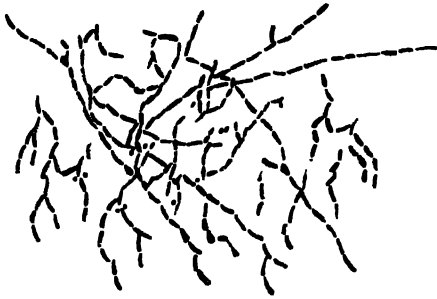
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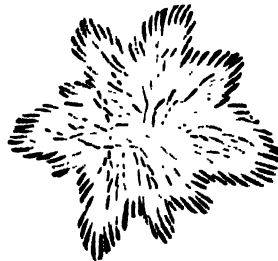
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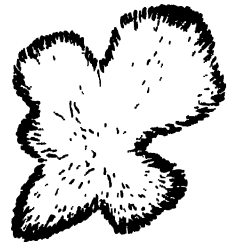
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9

1, Hair in trichophytosis. 2, Hair in microsporosis. 3, Hair in kerion. 4, Hair in favus. 5, Tinea circinata. 6, Microsporon furfur. 7, Microsporon minutissimum. 8, Actinomyces. 9, Fungus of madura foot.



## RINGWORM

hair is attacked from below upwards and the cuticle remains intact.

**Trichophyton endothrix.**—The endothrix trichophytions, in which the fungus is confined to the hair-shaft, are mainly of human origin; they chiefly attack the scalp, but occasionally give rise to ringworm of the glabrous skin, beard, and nails. They are divided, according to differences in the form or colour of their cultures, into three chief types—(1) *Tr. crateriforme*, (a) white or cream-coloured crater-like cultures, (b) yellow or primrose crater-like cultures; (2) *Tr. acuminatum*, yellowish acuminate cultures; and (3) *Tr. violaceum*, violet cultures. The first variety gives rise to the majority of cases of trichophytosis of the scalp and body and a few nail cases, the second affects the scalp only, and the third accounts for a few scalp and beard and nail ringworms. There is also a group, called by Sabouraud "neo-endothrix," which produces cerebriform cultures.

**Trichophyton endo-ectothrix** (or, since the hair is first attacked from the outside, *ecto-endothrix*).—The fungus proliferates both outside and within the hair in the form of short mycelial chains running for the most part parallel to the shaft as in the endothrix variety, but sometimes also crossing it in a transverse or oblique direction.

These trichophytic fungi are almost entirely of animal origin, and give rise to inflammatory and suppurative lesions and to the condition known as *kerion*. Sabouraud divides them into those with small spores (*microïdes*) and those with large spores (*mégaspores*), and distinguishes numerous species according to their different cultural characters. For practical purposes, four main varieties may be described as occurring in this country:—

1. A cat ringworm (*Tr. felineum*), giving white cultures with rayed margins, and producing vesicular rings on the trunk and limbs of children or adults.
2. A horse ringworm (*Tr. equinum*), producing yellow cultures with marginal rays, and giving rise to scaly rings in the human subject.
3. A horse or cattle ringworm (*Tr. gypsum*), with luxuriant white plaster cultures, causing inflammatory and suppurative lesions of the skin or beard.
4. A bird ringworm giving rose-coloured cultures (*Tr. rosaceum*), and communicable to man as beard or body ringworm.

In connexion with deep-seated suppurative ringworms a lichenoid trichophytide, due to absorption from the local lesion, has been described (Jadassohn), and other observers have found that an extract of ringworm cultures can cause a cuti-reaction and can set up a certain degree of immunity after inoculation.

**Epidermophytosis.**—This is due to the *Epidermophyton inguinale* or *cruris*, which does not attack the hair, and causes eczematoid ringworm of the groin, axillæ, feet, hands, and other parts (tinea cruris or tinea marginata, formerly known as eczema marginatum and often called dhobie's itch). It yields yellowish powdery cultures on Sabouraud's proof agar.

*Tinea imbricata* is a tropical species of ringworm not met with in this country, which gives rise to scaly concentric rings spreading over the trunk. Three varieties have been described. Other tropical varieties of ringworm are tinea alba, caused by *Tr. purpureum* (*Epidermophyton rubrum*), tinea albigena by *Tr. albicans*, tinea tropicalis by *Tr. blanchardi* (Sabouraud), tinea nigro-circinata by *T. ceylonese*, tinea intersepta, due to *Endodermophyton castellanii*, and *Acladiosis*, an ulcerative dermatomycosis caused by *Acladium castellanii*.

**Symptomatology and diagnosis.**—Ordinary **microsporiasis of the scalp** is characterized by scaly, more or less circular, semi-bald patches on the scalp, varying in size and number according to the length of duration and extent of the infection. They are distinguished from those of *seborrhœa* and *psoriasis* by the short, broken, twisted hairs, which are enveloped in a greyish-white sheath and show under the microscope a swollen opaque shaft and a mosaic of closely-packed spores surrounding it. *Alopecia areata* presents smooth bald patches without scales, and short needle-shaped hairs without a sheath. *Favus* is characterized by the presence of yellow cups, a mousey odour, and scarring of the scalp. *Lupus erythematosus* sometimes gives rise to scars on the scalp, but they are generally few in number, irregular in shape, shiny and discoloured, and accompanied by lesions on the face or elsewhere.

**Tinea circinata** may accompany microsporiasis of the scalp in the shape of small discoid or ringed patches, and is then probably due to one of the animal microsporons, but in the great majority of cases ringworm of the body is caused by endothrix or ecto-endothrix trichophytions, giving rise to scaly rings or, in the latter case, to inflammatory vesicular or



## RINGWORM

pustular circinate patches. These lesions have a well-defined vesicular or pustular margin distinguishing them from patches of *eczema* or *psoriasis* or the primary patch of *pityriasis rosea*.

**Trichophytosis of the scalp**, if due to the endothrix fungus, generally takes the form of small disseminated patches. The diseased hairs have no spore sheath, and are sometimes broken off on a level with the scalp, constituting the so-called "black dot" ringworm; or the patches may be "bald ringworm." The ecto-endothrix fungus gives rise to the more inflammatory types of ringworm, especially the type known as *kerion*, although *kerion* may also be caused by animal microsporons or by the endothrix fungus. The aggregation of suppurating follicles forms large soft prominent masses in which are buried the diseased hairs. A few cases of trichophytic granulomata have been described.

**Epidermophytosis.**—The fungus does not attack the scalp, but causes large red eczema-like patches with well-defined spreading margins in the groins (dhobie's itch) or axillæ, and may also give rise to vesicular or scaly patches on the hands or feet, white macerated patches between the toes, or thick scaly patches on the palms and soles.

**Ringworm of the beard.**—*Tinea barbæ*, or *tinea sycosis* as it is sometimes called, is common amongst those whose work brings them into contact with horses and cattle. In other cases the barber's brush is the vehicle of infection. It may appear as an erythematous ring enlarging peripherally, or in the form of scattered scaly follicular papules or pustules. In some cases in an advanced stage there are inflammatory nodular masses analogous to *kerion* of the scalp. Careful examination will reveal the presence of short, greyish, broken hairs which show under the microscope the mycelial chains outside and inside the hair characteristic of the endo-ectothrix fungus, and serve to distinguish the disease from *staphylococcic sycosis*. Exceptionally, the endothrix variety is found. Apart from microscopical examination, the diagnosis from *staphylococcic sycosis* is often difficult. *Eczema* and *impetigo* are superficial and not localized to the hair-follicles. The hair of the upper lip is rarely attacked in *tinea sycosis*.

**Ringworm of the nails** is caused by one of the trichophytions, the ecto-endothrix or endothrix (*Tr. acuminatum* or *Tr. violaceum*), or by the epidermophyton. The nails become discoloured,

friable, and partially disintegrated. *Eczema*, *psoriasis*, and infection of the nails by cocci are generally associated with other lesions characteristic of these diseases, and the presence of mycelium in the scrapings, when examined under the microscope, confirms the diagnosis of ringworm. *Favus* can be distinguished by the special characters of the mycelium and by cultures.

**Prognosis and treatment.**—The treatment of *tinea tonsurans* by ointments and lotions is notoriously long and tedious. A few types, such as *kerion* and bald ringworm, may recover spontaneously, but the disease generally lasts six to eighteen months and often longer. Strong antiparasitic ointments, such as those containing chrysarobin, croton oil, iodine, mercury, sulphur, carbolic acid, salicylic acid, copper, picric acid, or sodium chloride, are employed. A great deal depends upon the care and perseverance devoted to their application. Those which set up a certain amount of inflammation in the hair-follicles are the most rapid in their action, and needling each hair with croton oil is a useful method for small patches or residual stumps. Frequent washing with soft soap, keeping the hair short so as to facilitate the application of the ointment, and the wearing of linen or paper caps to prevent infection are important items in the treatment. The best method of dealing with ringworm of the scalp consists of epilation by means of the X-rays, although the method is not free from risk and must be conducted by an expert. In the majority of cases it is advisable to depilate the whole scalp, since partial epilation is almost certain to be followed by reinfection of the healthy parts or by the discovery of diseased hairs which had been previously overlooked. Adamson's method is the best for this purpose. Three converging pegs are fitted around the aperture in the box or shield containing the tube, so that the patient's head is maintained in the correct position and at the correct distance from the antikathode. Five points are then marked on the scalp with an aniline pencil in the frontal, vertex, occipital, and inferior parietal regions respectively, each being 5 in. apart. Each of these points forms a centre to which the ends of the pegs converge as they rest on the scalp. The exposures overlap, so that the whole of the scalp receives an equal dose, and the scalp is epilated in five applications. The hair begins to fall in about a fortnight, and the whole scalp is completely denuded of hair in three weeks, and is then free

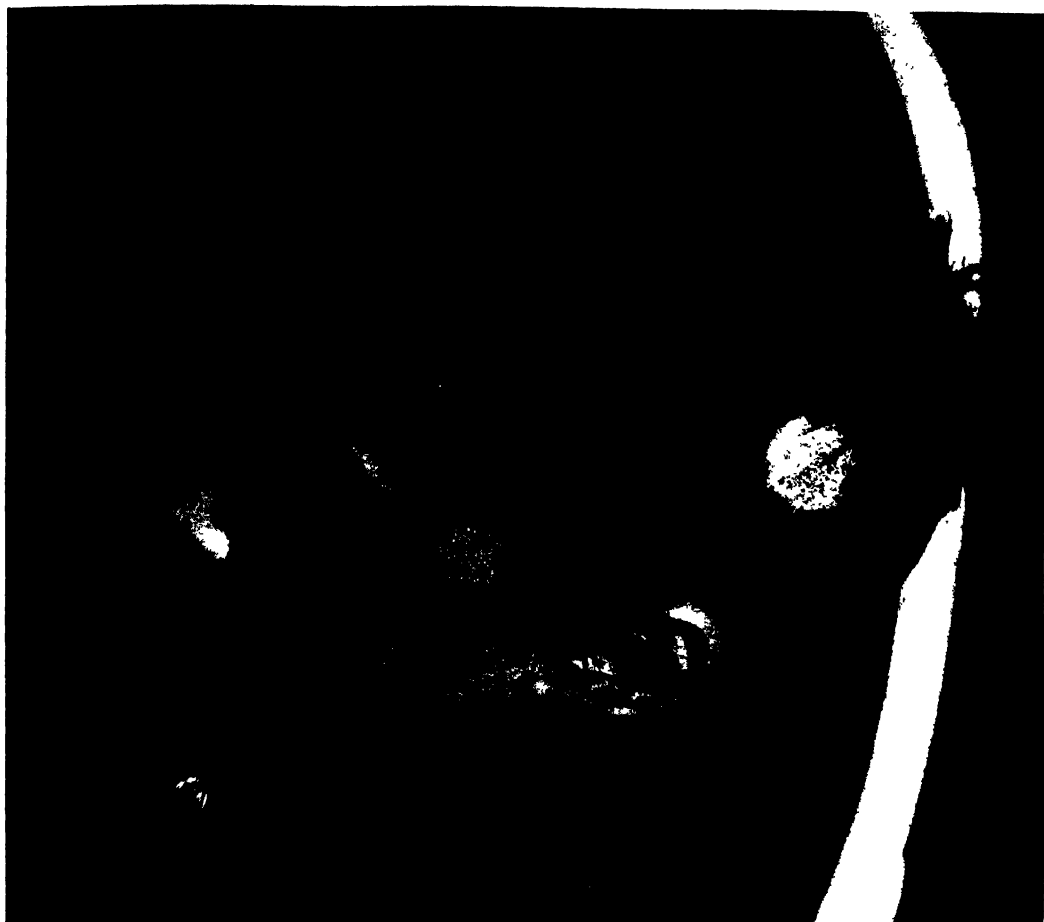


PLATE 29.—TINEA TONSURANS AND SUPPURATING TINEA CIRCINATA  
OF ARM.



## ROCKY MOUNTAINS FEVER

from infection. Regrowth begins in six weeks and is complete in about three months. While the hair is falling the scalp should be washed frequently and a mild antiseptic ointment applied.

**Tinea circinata** can be removed in a few days by painting the patches with strong solution of iodine or by applying mercurial or sulphur ointments. **Tinea marginata** is more refractory, and ung. chrysarobini is often used for this variety of ringworm, but a less irritant and equally efficacious application is an ointment consisting of benzoic acid and salicylic acid, about 20 gr. of the former and 15 gr. of the latter, to an ounce of vaselin and coconut oil (Whitfield).

The treatment of **ringworm of the beard** consists in epilation by X-rays, which can be accomplished by a method similar to that employed in ringworm of the scalp. Failing the X-rays, epilation by forceps and the application of mild antiseptics such as a weak solution of perchloride of mercury and the dilute nitrate-of-mercury ointment may be used, but with this treatment the disease is apt to be very chronic. For ringworm of the nails complete ablation is the most satisfactory method.

S. F. DORE.

### BITTER'S DISEASE (see IMPETIGO CONTAGIOSA).

**ROCKY MOUNTAINS FEVER** (*syn.* Spotted Fever of the Rocky Mountains, Black Fever, Blue Disease).—A specific fever resembling typhus in its symptoms, and occurring among the foot-hills of the mountains in several of the western of the United States. It follows the bite of a tick—*Dermacentor venustus* (PLATE 38, Fig. 6, facing p. 366), and is due to small bodies resembling, but somewhat larger than, the Rickettsia bodies found in lice fed on patients affected by typhus or by trench fever. The disease is most prevalent in April, May and June, and attacks individuals of any age and either sex.

**Symptoms.**—A period of malaise is followed by chills, which recur irregularly and in diminishing severity throughout the illness. The temperature gradually rises, reaching 103° or 104° F. by the second day and 105° or higher by the fifth. Pains in the joints are severe. Toxæmia is evident, prostration, with delirium and partial or complete loss of consciousness, developing rapidly, so that a typhoid-like state is present. The eruption appears from the

fourth to the seventh day on the wrists, ankles or back, and rapidly spreads, to become generalized, not sparing the scalp, hands or feet. At first there are discrete rose-coloured spots, which soon become petechial and enlarge; they may be confluent, especially on the more dependent parts. The speckled rash when the spots remain discrete has given rise to the name of "spotted fever," and their dark colour to those of "black fever" and "blue disease."

Slight jaundice may be present. The spleen is considerably enlarged and is tender, while the liver enlarges to a less degree. Constipation is the rule. The urine is diminished, and may contain albumin and casts. In severe cases œdema of the face and limbs occurs early. Towards the beginning of the second week nausea and vomiting appear, and may continue to the end in fatal cases. The pulse and respiration-rate are quick. Slight catarrh of the respiratory tract is found. The blood shows little change, the red cells and hæmoglobin being slightly reduced and the leucocytes slightly increased. In cases which recover, the fever begins to subside by lysis at the end of the second week, defervescence being accompanied by desquamation. Gangrene may occur in the skin in places where it is tight, as the ears, toes, fingers, and elbows, or elsewhere as in the tonsils, scrotum, or prepuce. The case-mortality is very variable, being as high as 90 per cent. in Montana and as low as 5 per cent. in Idaho.

**Morbid anatomy.**—Post mortem, in addition to congestion of the viscera, the myocardium is found to be soft and the spleen and the glands large and firm; the liver shows fatty degeneration. There are subserous hæmorrhages, and others into the genitalia.

**Treatment.** Prophylaxis is concerned with eradicating the infecting tick. Its larval and nymph stages develop principally on the ground squirrel and the woodchuck, and attempts are being made to exterminate these animals in infected districts. The adult tick, which bites man, is harboured by the Rocky Mountain goat, the sheep, brown bear, coyote, badger, and wild cat. Goats and sheep are being dipped to destroy the ticks and prevent their dissemination. The treatment of the developed disease is symptomatic.

FREDERICK LANGMEAD.

### RODENT ULCER (see SKIN, MALIGNANT GROWTHS OF).

## RUBELLA

**RÖNTGEN RAYS** (see X-RAYS).

**ROSACEA** (see ACNE ROSACEA).

**RÖTHELN** (see RUBECLA).

**RUBELLA** (*syn.* German Measles, Rötheln).—An acute contagious disease characterized by a polymorphous eruption, enlargement of the superficial lymphatic glands, and an habitually mild course.

**Etiology.**—The existence of rubella as a morbid entity distinct from scarlet fever and from measles is now well established. Rubella always breeds true. An attack of scarlet fever or measles is no protection against rubella, nor does rubella protect against scarlet fever or measles. On the other hand, a second attack of rubella is extremely rare.

The causal agent has not yet been discovered. A. F. Hess has examined the blood bacteriologically during the height of the eruption without obtaining a growth in any case. He also found that intraperitoneal injections of the blood into *Macacus rhesus* monkeys gave a negative result.

Direct infection is chiefly responsible for transmission of the disease; the possibility of indirect infection by third persons or fomites is doubtful. The infectivity of rubella is highest in the prodromal and eruptive stages, and ceases rapidly after the disappearance of the rash.

The disease is uncommon in infancy and most frequent between the ages of 5 and 15. Adults are more susceptible to it than to measles, from which most have already suffered. It is rare after 50. Both sexes are equally liable. The morbidity is greatest in the winter and spring, or spring and early summer.

**Symptomatology.**—The incubation period is rarely less than 10 or more than 21 days; it is usually 15 or 16 days.

**Premonitory symptoms** are usually absent, and the rash is the first manifestation of the disease, but occasionally a prodromal period of two to five days occurs in which the patient, especially if an adult, may complain of headache, backache, muscular fatigue and loss of appetite, and during which the temperature is slightly raised. Stiffness of the neck and enlargement of the cervical and occipital glands, as well as conjunctivitis or an injection of the conjunctival vessels ("pink eye"), may precede the eruption. Prodromal erythematous and urticarial rashes have been observed.

The eruption first appears upon the face,

where, unlike scarlet fever, it invades the circumoral region and then rapidly spreads over the trunk and limbs. It consists of pale pink papules, round or irregular in shape, ranging in size from a pin's head to a split pea. The lesions are usually most profuse on the outer surface of the thighs and buttocks. By the time the rash is well marked on the trunk and lower limbs, it has faded on the face and is disappearing on the chest. It is often accompanied by itching. As in scarlet fever and measles, miliary vesicles and petechiæ may accompany the specific eruption.

The individual lesions are sometimes large, blotchy, and crescentic, and closely resemble those of measles (*rubella morbilliformis*). In *rubella scarlatiniformis*, which is less frequent, the rash on the trunk closely resembles scarlet fever, from which it differs chiefly by its distribution on the face, where it invades the circumoral region. Not infrequently the morbilliform and scarlatiniform types coexist in the same patient. The eruption lasts from two to five days. As it fades it leaves a purplish or mottled appearance of the skin. Desquamation is usually fine and branny, and may be absent. Relapses are rare.

Catarrhal signs are the rule, but are much less pronounced than in measles. Conjunctivitis is common, but is usually of very mild degree. There may be a watery nasal discharge and coughing and sneezing are not infrequent.

The buccal mucous membrane may show some injection and even punctiform hæmorrhages, but Koplik's spots are never present. Forstheimer has described as the characteristic enanthem of rubella an eruption of small discrete dark-red spots on the soft palate and uvula.

**Temperature.**—In a large number of cases the temperature does not rise above 99° F., and is often not raised at all. Constitutional disturbance is slight or absent. Temperatures above 102° F. are most exceptional. The duration of the pyrexia is two or three days.

**Lymphatic glands.**—Enlargement of the posterior cervical glands is a constant feature. The occipital, submaxillary, axillary, and inguinal glands are involved to a less extent. The swollen glands are seldom painful and only slightly tender, and soon resume their normal condition after disappearance of the rash. Suppuration is most exceptional. Slight enlargement of the spleen occasionally occurs.

**Blood.**—Leucopenia with relative lymphocytosis is characteristic. Lymphocytosis may occur before the appearance of the eruption,

## RUBELLA

and thus help to separate infected from non-infected children (Hess). According to Saito, there is an increase in the number of neutrophils at the height of the eruption, and eosinophilia is frequent when the eruption is fading.

**Aberrant forms.**—Cheadle and others have described a severe form of rubella characterized by high temperature, rapid pulse, continual nausea and vomiting, diarrhoea, tonsillitis, laryngitis, convulsions, and delirium. Such cases are extremely rare, and it is highly probable that most of them were really examples of scarlet fever or measles.

As in the other acute exanthems, a non-eruptive variety of rubella has been described, in which the skin eruption is absent, but there is an enanthem on the mouth and palate. The existence of such forms, in the absence of bacteriological confirmation, which in the present state of knowledge is impossible, is, of course, extremely doubtful.

**Association with other diseases.**—Rubella may develop during the incubation period of, or during convalescence from, one of the other acute infections, but the association is not frequent.

**Complications.**—As a rule, the disease runs its course without any complications, and it is extremely probable that in some at least of the complications attributed to rubella an erroneous diagnosis has been made, the real disease being scarlet fever, measles, or an association of the two. This particularly applies to the secondary cases of rheumatism, nephritis, otitis, tonsillitis, bronchitis, broncho-pneumonia and pleurisy that have been attributed to rubella.

Nervous complications are very uncommon, but Ker states that adult patients often complain of neuralgia and toothache, which he attributes to pressure on the nerves by the enlarged glands.

**Diagnosis.**—Rubella is most likely to be confounded with scarlet fever and with measles, and such mistakes in diagnosis are very frequent. The onset of *scarlet fever* is usually more violent. Headache and sore throat, though occasionally present in rubella, are seldom intense, and vomiting, so constant as a prodromal symptom of scarlet fever, is rare in rubella. A normal or subfebrile temperature on the appearance of a scarlatiniform eruption points to rubella rather than scarlet fever.

The scarlatiniform variety of rubella closely simulates scarlet fever, but the distribution on the face, where it invades the zone of circum-oral pallor, is characteristic. When the rash has faded from the face, the diagnosis of rubella

from the eruption alone may be impossible, and other circumstances must be considered, such as the history of infection, the character of the onset, the subsequent desquamation and development of complications peculiar to scarlet fever, such as rheumatism or nephritis.

Rubella is distinguished from *measles* by the absence of Koplik's spots, the paucity of the catarrhal signs, and the appearance of the eruption on the first day of the illness.

The tuberculin cuti-reaction may help in the diagnosis of rubella from measles, but only in tuberculous children. A positive reaction in the eruptive stage excludes measles. On the other hand, a negative reaction is no proof of measles, since, in the absence of tuberculosis, the reaction is negative in rubella also.

The enlargement of the posterior cervical glands is usually more marked in rubella than in either scarlet fever or measles, and the diazo reaction, which is almost invariable in measles even for a few days after the disappearance of the eruption, and only slightly less frequent in scarlet fever, is rarely positive in rubella, and then only during the eruptive period.

Late *serum rashes* on their first appearance may sometimes simulate rubella, and the resemblance is increased by the frequent association with cervical adenitis; but their subsequent development, when they usually assume a circinate appearance, and the frequent coexistence of pains in the joints and muscles, are distinctive. Rashes caused by certain drugs, especially *copaiba*, may be confounded with rubella, but cervical adenitis is absent. The roseola of *sypilis*, though accompanied by generalized adenitis, always spares the face and is associated with other secondary symptoms, and usually with the remains of the primary lesion.

**Prognosis.**—The prognosis is extremely good. Out of 2,572 cases admitted to the hospitals of the M.A.B. in a period of fourteen years, only two deaths occurred, one of which was due to general tuberculosis.

**Treatment.**—No special treatment is required. The patient should be kept in bed during the eruptive stage, but may be allowed up at the end of a week if the diagnosis is certain.

J. D. ROLLESTON.

**RUBEOLA** (*see* MEASLES).

**RUMINATION** (*see* STOMACH, FUNCTIONAL DISORDERS OF).

**RUPTURE** (*see* HERNIA, ABDOMINAL).

**SACHS' DISEASE** (*see* IDIOCY, AMAUROTIC FAMILY).

**SAORAL PLEXUS, LESIONS OF** (*see* SPINAL NERVES, LESIONS OF).

**SACRO-ILIAO DISEASE** (*see* ARTHRITIS, TUBERCULOUS).

**SALIVA, ANOMALIES OF SECRETION OF.**—The normal amount of saliva secreted in twenty-four hours has been estimated at two to three pints. Its flow is temporarily increased by the sight of food, the presence of dry, irritating particles or ulceration in the mouth, or as a precursor of vomiting; it may be temporarily decreased by emotion, such as anger or fear.

**Ptyalism** is the name given to a pathological and more lasting excess of secretion than that due to the causes mentioned above. It is a common result of excessive doses of certain drugs, notably pilocarpine, iodides, and mercury. In patients who show an idiosyncrasy to mercury, profuse salivation has followed the administration of 10 doses of  $\frac{1}{2}$  grain of calomel. Such an idiosyncrasy is not very uncommon, and its possible presence necessitates care in the administration of mercurial drugs to a patient for the first time. When mercurial salivation occurs it may continue for three weeks after the drug is discontinued. Ptyalism is sometimes seen in a complex group of conditions, including *chronic pancreatic disease*, in which its presence may be a helpful indication in forming the diagnosis; *pregnancy* in the early months, often, but not always, associated with morning vomiting; *pelvic disease*, especially uterine disorders about the time of the menopause, and *hysteria* in young subjects. Hysterical ptyalism commences suddenly, may be confined to one gland, is often associated with other hysterical manifestations, may resist treatment for a long time, but ultimately ceases as suddenly as it commenced. *Bulbar palsy* may be a cause of severe ptyalism. In *hydrophobia* there is a great flow of viscid saliva. Profuse salivation is occasionally seen as an early symptom of *general paralysis of the insane*.

The **treatment** of ptyalism consists in removal of the cause if possible and the administration of belladonna in large doses, accompanied by the use of astringent mouth-washes and general "tonic" measures.

**Xerostoma**, or dry mouth, is a condition in which the salivary secretion is extremely deficient or even absent. It affects chiefly elderly females of a neurotic temperament, and may be very resistant to treatment. The condition is probably atrophic, and a similar process in nose and conjunctiva may be coexistent. The lack of secretion gives rise to dryness of the mouth, furring of the tongue, and fissures of the buccal mucous membrane, with consequent loss of appreciation or dislike of food, leading to impairment of the general health.

**Treatment** is difficult, and must be active, including the very frequent use of mild antiseptic mouth washes, very careful attention to the hygiene of the mouth, and the administration of fairly large doses of jaborandi or pilocarpine. Some mild cases which have resisted medicinal treatment have yielded to "suggestion," a result which might be expected in view of the frequent presence of a neurotic element. In a certain proportion of cases the condition may be regarded as a permanent abnormality analogous to scleroderma with deficiency of sweat secretion.

C. E. SUNDELL.

**SALIVARY CALCULI.**—These concretions occasionally occur in the substance or in the main ducts of the salivary glands. In the former situation they may give rise to no symptoms; in the latter they may be felt by the patient as a hard substance in the cheek or under the tongue. They are composed for the most part of phosphates or carbonates; in some a nucleus of degenerated epithelium or a foreign body exists. It is probable that they owe their origin to past catarrhal conditions of the epithelium of the gland or duct. The severity of the symptoms to which they give rise depends upon their size and the occurrence of secondary inflammatory changes. A calculus of moderate size may produce complete obstruction of the duct with consequent swelling and distension of the corresponding gland, leading to acute inflammation or chronic enlargement, with hypertrophy of the interstitial tissue. Sometimes the obstruction and consequent swelling of the gland are intermittent in character.

**Treatment.**—An impacted calculus should, if possible, be coaxed along the duct, but if necessary an incision should be made through

## SALPINGO-OÖPHORITIS

the buccal mucous membrane and the calculus removed. The formation of an intra-buccal salivary fistula is of no importance, but an external fistula consequent upon abscess-formation and ulceration through the cheek causes great inconvenience and may be very slow in healing.

C. E. SUNDELL.

### SALIVARY GLANDS, INFLAMMATION OF (see PAROTITIS; MUMPS).

**SALPINGO-OÖPHORITIS.**—Inflammation of the tube and ovary by which they are usually bound together by peritoneal adhesions.

**Etiology.**—Salpingo-oöphoritis is always the result of infection, which, in the great majority of cases, ascends from the uterus to the tubes and then to the peritoneum and ovary. Sometimes the inflammation comes from a source in the abdominal cavity. Rarely blood infections are responsible.

Gonorrhœa and puerperal infections account for most of the cases. When a woman acquires gonorrhœa the infection often remains limited to the cervical canal and to the parts below it, but in many cases the organisms steadily increase and, invading the endometrium, ultimately reach the mucous membrane of the tube and the peritoneum. When the disease becomes chronic and limited above by the internal os, it may again be lighted up by some disturbance of the cervix, and extension to the uterus, tubes, and the peritoneum may then occur. Dilatation of the cervix by the delivery of a child or the expulsion of an abortion is a very common cause of the extension, and in these circumstances the tubal inflammation may appear to arise from puerperal infection when in reality it is due to extension of a pre-existing gonorrhœa. Sometimes extension occurs without obvious cause.

*Infection of the puerperal uterus* by the streptococcus is a common cause of salpingitis and consequently of oöphoritis. The tube may be invaded by spread of the infection directly from the uterus, just as in the case of gonorrhœa. It may also be infected from the placental site, the inflammation spreading along its veins between the layers of the broad ligament and there setting up a cellulitis from which the infection traverses the wall of the tube.

Infection from above may be tuberculous or from the bowel, especially the appendix. Cases occur fairly often in which the appen-

dix and right appendages are matted together in one inflammatory mass, due to chronic appendicitis.

**Pathology.**—The tube becomes engorged with blood and swollen, its mucous membrane and fimbriæ red and œdematous, and its peritoneum inflamed, with engorged blood-vessels on its surface, of a pink or red colour. Microscopically, a section of the tube shows swelling and engorgement of the mucous membrane with round-celled infiltration of the stroma.

Acute inflammation of the tube may subside completely, but usually recovery is incomplete and the disease becomes chronic. Acute attacks then recur from time to time and produce characteristic changes in the tube-wall, the infiltration being gradually increased and the wall becoming thickened in all its layers.

The lumen of the tube at the uterine end is narrow, and is quickly occluded by the swelling of its mucous membrane; infected discharge from the tube therefore finds its way through the abdominal ostium into the peritoneal cavity and produces peritonitis around the end of the tube. Infection may also cause peritonitis by traversing the wall of the tube. The abdominal ostium of the tube frequently becomes closed by peritoneal adhesions or by its open end becoming adherent to the ovary or some surface covered by peritoneum. The occlusion may be caused by the edges of the fimbriæ, which are covered by peritoneum, becoming adherent to one another; when this occurs the fimbriæ are turned inwards and form a tuft of mucous membrane inside the tube surrounding the closed ostium. No matter how the tube is closed, there is a strong probability of the ovary being involved in the inflammatory process. The ostium being closed, if the infection is sufficiently virulent pus collects in the tube, usually in its ampullary portion, and forms a *pyo-salpinx*. In this event there is, as a rule, extensive pelvic peritonitis, matting the tube and ovary together in such a way that it is often difficult to distinguish the one from the other until after the pyo-salpinx has been removed and opened.

When the abdominal ostium remains open or is fixed to the ovary, intraperitoneal collections of pus often form near the end of the tube and in contact with the ovary. Inflammation of the ovary is almost inevitable when it is lying in contact with and adherent to a pyo-salpinx, or when an abscess forms around the fimbriated end of the tube; it is found



## SALPINGO-OÖPHORITIS

also in some degree in all cases in which the tubes are subject to recurring acute attacks of inflammation.

**Oöphoritis.**—Inflammation of the ovary is an extremely rare occurrence except in association with and secondary to infection from the tube or intestine.

In rare instances it complicates general infections such as mumps and scarlet fever. Tubal inflammation is the cause of the great majority. In acute gonorrhœal salpingitis the ovary becomes engorged and œdematous, but in the early stage is probably not actually infected. In virulent puerperal cases, on the other hand, very rapid infection of the ovary may ensue, the gland becoming œdematous and covered with lymph and small abscesses developing in it.

In chronic oöphoritis the ovary is enlarged, its capsule thickened, and its surface often roughened from adhesions and studded by numerous distended follicles. An abscess in the ovary is a common sequel to salpingitis, and usually arises by direct infection from the tube of a follicle or corpus luteum; such an abscess may run a subacute or chronic course. Should the wall between the abscess in the ovary and the lumen of the tube break down, a tubo-ovarian abscess is formed.

**Symptomatology.**—The *acute* symptoms are generally due to the peritonitis set up by the initial attack or by an acute exacerbation in a chronic case.

An attack is marked by severe abdominal pain with vomiting, raised temperature, and accelerated pulse-rate. In the course of a few hours the pain usually becomes localized to the lower part of the abdomen, which is rigid and tender and moves poorly on respiration. Later the tongue is furred and the patient disinclined for food; there is usually constipation, and sometimes pain on micturition.

The infection producing an acute attack may be virulent enough to cause a rapid general infection of the peritoneum. Fortunately, the cases in which the peritoneum is unable to localize the infection are comparatively rare; when they occur there has usually been no previous attack, and consequently it is often impossible to decide the cause of the peritonitis. General peritonitis may also result from rupture of a pyo-salpinx, but this is still less common.

Symptoms of peritonitis limited to the pelvis and recurring from time to time are the most usual evidence of salpingo-oöphoritis. It is

common for an increase in vaginal discharge to be noticed during the attacks.

In *chronic* cases there is often a history of ill-health, loss of weight, pelvic pain and backache, dating from an attack of gonorrhœa, or from a labour or miscarriage followed by an abnormal lying-in. In the absence of definite information about the puerperium, the length of time that the patient was kept in bed is a valuable guide to her progress after labour.

Recurring attacks of pelvic inflammation at intervals that may vary from a few weeks to many months are the rule; they may be so mild that the patient can lead her usual life, or severe enough to keep her in bed for two or three weeks at a time. Sterility due to the occlusion of the tubes is a very common sequel, and is more likely to follow gonorrhœa, which usually attacks both tubes, than puerperal infection, in which the salpingitis is often unilateral.

Since the ovary and inflamed tube are frequently bound down to the floor of Douglas's pouch by peritoneal adhesions, coitus may be painful. When the uterus is retroverted and fixed down on top of the appendages, some pain high up in the vagina is almost inevitable during sexual intercourse.

Dysmenorrhœa frequently results from engorgement of the pelvic organs, and is usually most severe when the uterus is fixed in a retroverted position. This position is often an additional cause also of severe backache and menorrhagia, though both are commonly associated with salpingo-oöphoritis apart from retroversion.

Menorrhagia is probably due chiefly to the infection of the endometrium, which in most cases is affected before the tube.

**Diagnosis.**—In the initial attack the physical signs are usually indefinite and the diagnosis must be made on a history of infection after labour, of gonorrhœa, or on such evidence as profuse discharge or warts on the vulva. There may be tenderness in the pelvis and lower abdomen, and obvious pulsation of the vessels in the fornices.

As the peritonitis becomes more localized, adhesions may shut off collections of peritoneal fluid round the tube and mat the gut and omentum in this position in such a way as to form a palpable mass.

When the symptoms are referred to the right side, in the absence of evidence of vaginal infection or previous inflammation of the

## SALPINGO-OÖPHORITIS

appendix it is often impossible to differentiate between *appendicitis* and *salpingitis*.

If the salpingo-oöphoritis has existed for some time, definite physical signs can usually be made out, especially when there have been recurring attacks.

The tube becomes thickened, hard and possibly distended, the ovary bulky, and there may be small encysted collections of peritoneal fluid or pus round and between the tube and ovary. The mass so produced can be detected on bimanual examination, and usually lies in the postero-lateral part of the pelvis or in Douglas's pouch immediately behind the cervix. It is fixed, or capable of only a small amount of movement, and is tender. Though usually hard, it is elastic if the appendages are surrounded by encysted fluid.

In some cases the mass is found to be fixed above the brim of the pelvis. When situated in this position it is usually the result of an attack of peritonitis during the puerperium while the appendages are still raised by the enlarged uterus. By becoming adherent to the abdominal wall or the iliac fossæ it cannot descend into the pelvis as involution proceeds.

**Prognosis.**—In the fulminating cases in which general infection of the peritoneum occurs the outlook is extremely grave; almost all the patients succumb.

After adhesions have developed and shut off the inflammatory focus, the risk to life is small.

There is, however, a great probability that the inflammation will become chronic, recurring attacks of acute inflammation succeeding each other at progressively shorter intervals. The patient is thus reduced to a state of invalidism, from which she can only be delivered by operation.

In some cases, which are usually mild, the inflammation resolves and the appendages are restored to the normal. In others, a few acute exacerbations follow the original attack at lengthening intervals and leave damaged and, very often, closed tubes, causing sterility but few, if any, other symptoms.

The results of operation are good in that cure of pain and acute attacks is to be expected. The risk of general peritoneal infection is not great if the operation is undertaken during a quiescent period, but it may involve the removal of both tubes and ovaries—a very serious drawback in the case of young women.

**Treatment.**—When *general peritonitis* is present the only hope of saving the patient is by laparotomy and drainage of the peritoneal cavity.

In *other acute cases*, whether the attack be an initial or a recurrent one, surgical treatment should be avoided whenever possible. Not only are the risks of operation very much reduced during a quiescent period but in many cases the acute symptoms, though they may be very severe, subside completely and operation may prove to be unnecessary. Absolute rest in bed is essential. Simple methods such as hot fomentations to the abdominal wall and small doses of brandy by the mouth should be employed for the relief of pain and sickness. Later, fluid diet should be ordered, and the aperients necessary to ensure a daily action of the bowels.

If with rest in bed the peritonitis becomes localized, there will probably be definite signs of improvement within a week or ten days, during which time the pain will steadily decrease and the temperature and the pulse-rate fall. The tenderness in the pelvis will diminish and the mass show signs of resolution.

Early surgical interference is needed when there is evidence that the peritonitis is spreading. It is also indicated when the pelvic mass increases in size, the temperature and pulse-rate remain high and the pain persists; in these circumstances it is probable that suppuration has occurred.

At this early stage laparotomy is frequently the only method of evacuating the pus and draining the cavity. Its great drawback is a serious danger of causing a further spread of the peritonitis, but, when it is necessary, this risk must be taken, as it is smaller than that of expectant treatment. Fortunately, in many cases when suppuration occurs the collection of pus is in Douglas's pouch and is accessible from the vagina by posterior colpotomy without opening the general peritoneal cavity. Unless there is a definite collection which bulges down the posterior vaginal wall this latter operation should not be attempted, as the gut may be adherent to the back of the uterus or the floor of Douglas's pouch and may be injured.

After a pelvic abscess has been drained by posterior colpotomy there is usually a striking improvement in the patient's condition within twenty-four hours, and three or four days later the tube can be removed without fear of the wound closing before the inflammation has subsided.

When a patient's condition is grave and the abdomen has to be opened during an acute attack, it is often unwise to attempt more than the drainage of an abscess or of the peritoneal

## SALPINGO-OÖPHORITIS

cavity, when the inflammation is not shut off by adhesions. If, however, the diseased appendages can be removed at the same time without great difficulty or undue disturbance, this is the better procedure; if the infected area is merely drained a sinus is likely to persist and add greatly to the difficulties of any subsequent operation.

In *chronic cases* the treatment may be medical or surgical. It is often difficult to decide which of the two is the better for any particular case, and if there is doubt, medical treatment should be tried first.

When there is much enlargement of the appendages, when the general health is much impaired, or when the recurring attacks of pelvic inflammation are frequent and severe—especially if the intervals between them are becoming shorter—the prospects of a cure, except by removal of the diseased appendages, are very remote.

If the attacks have been mild and few in number and there are no obvious physical signs on bimanual examination, treatment by medical means may be expected to suffice.

In the moderately severe cases the results of medical treatment are more uncertain, but all patients, and especially those in the child-bearing period of life, should be encouraged to give it a thorough trial.

A patient undergoing medical treatment for salpingo-oöphoritis must be prepared to avoid fatigue and exposure to cold and to lead a quiet life, perhaps for many months. Among women of the working classes this is often impossible, and many others are unwilling to submit to a long period of inactivity if, after all, the result may be disappointing.

Surgical measures, therefore, often have to be undertaken when prolonged treatment by medical means might effect a cure.

**Medical treatment.**—In mild cases douching and limitation of exercise often give excellent results. The patient may be allowed to be up and about, but should avoid sexual intercourse, risk of chill, much walking or standing, and all unnecessary exertion; the bowels should be carefully regulated, by a daily aperient if necessary, and two large hot douches should be given daily. The value of douches in promoting resolution of pelvic inflammation depends on their temperature and volume; each douche should amount to four or five pints and be given as hot as the patient can comfortably bear it.

If this treatment fails to give relief, a course

of glycerin tampons or medicated pessaries should be tried for ten days or a fortnight. During this time the patient should remain in bed, a tampon or pessary being introduced each day and left in the vagina for eight or ten hours; a large hot douche should be given before the pessary is put in and after it is removed.

The pessaries consist of a medium, melting at body temperature, in which various drugs are incorporated; glycerin, ichthyol, and iodine are the most useful.

Such a course may be repeated two or three times if required at intervals of a few weeks, and douching should be continued between the courses.

At many of the spas in this country and on the Continent special treatments by baths, rest, radiant heat, and douching are carried out for pelvic inflammation, and in obstinate cases should be tried whenever possible. Drugs given by the mouth have very little effect, but mercury and iodide of potassium seem sometimes to have a favourable influence, even when there is no evidence of syphilis.

**Surgical treatment.**—Removal of the diseased appendages by abdominal section is the only method of dealing with cases of chronic salpingo-oöphoritis when medical treatment has failed. When the disease is bilateral it may be necessary to remove both appendages, but every effort should be made to save some ovarian tissue, particularly in young women. Where there is still any prospect of pregnancy occurring and there seems a reasonable chance of one tube recovering or of its being made patent by salpingostomy, it is better to leave it and risk a second operation than to remove it and make further pregnancy impossible. In women after the late thirties when both tubes or both appendages have to be sacrificed, it is advisable to remove the uterus and cervix at the same time. This operation can be done without appreciably increasing the risks, and has the advantage of curing the vaginal discharge which is commonly present and of preventing the severe uterine bleeding which is likely to occur at the menopause when the uterus has been infected.

In young women in whom it is possible to save an ovary or part of an ovary, the uterus should not be removed unless there is menorrhagia or marked gonorrhoeal discharge.

Drainage is not often necessary in chronic cases, but when it is, total hysterectomy provides for it.

J. P. HEDLEY.

## SCABIES

**SALVARSAN, TOXIC EFFECTS OF** (see SYPHILIS).

**SANATORIUM TREATMENT** (see TUBERCULOSIS FROM THE STANDPOINT OF PREVENTIVE MEDICINE; PULMONARY TUBERCULOSIS).

**SAPONIFICATION** (see POST-MORTEM EXAMINATIONS IN MEDICO-LEGAL CASES).

**SAPRÆMIA, PUERPERAL** (see PUERPERAL FEVER).

**SARCOMA** (see under individual organs).

**SCABIES.**—An itching disease of the skin caused by a small mite, the *Acarus* (or *Sarcoptes*) *scabiei* (PLATE 24, Fig. 4, Vol. II, facing p. 487).

**Etiology and pathology.**—The disease may occur at any age and in either sex. It is most common among the poorer classes, and is contracted by direct contact or, less often, from the clothes or bed linen of infected persons. The infection spreads from one person to another through families, schools, lodging-houses, etc. Children at the breast frequently contract the disease from close contact with their mothers. Sexual intercourse is also a common cause of infection. In rare cases scabies is of animal origin, dogs, horses, or cats being the usual offenders. There is also an uncommon and severe form met with in Norway in which the acarus is said to be derived from the wolf.

The acarus is oval or nearly round in shape, the female measuring 330 to 450  $\mu$  in length by 250 to 350  $\mu$  in breadth, and the male 200 to 235  $\mu$  by 145 to 190  $\mu$  (Shipley). The mite possesses two anterior and two posterior pairs of legs; in the female the anterior pair end in pedunculated suckers and the posterior pair in long bristles, while the male is distinguished by having suckers on the hindmost pair of legs. The male is rarely seen, not only because of his small size but because he lives on the surface of the skin and is seldom caught. The female burrows into the horny layer and can be extracted by a pin thrust into the tunnel which she makes for the purpose of depositing her eggs, and is visible to the naked eye as a minute grey pearly object about half the size of a pin's head. The burrow is seen on the skin as a grey or black sinuous dotted line, from a millimetre to a centimetre in length. The acarus is found at the distal end, where there is often a vesicle. Eggs to the number

of one or more a day are deposited along the tunnel behind the acarus, since she cannot move backwards on account of her projecting bristles. The young six-legged acarus is hatched out in from three to six days, and is full grown in about a month.

**Symptomatology.**—There is intense itching, which is worse at night. Burrows and minute vesicles may be detected on the sides of the fingers and on the ulnar surface of the hands, the front of the wrists, the anterior axillary folds, on the penis, the nipples in women, in the umbilicus, on the elbows and ankles, between the toes and on the soles of the feet. There are also secondary lesions due to scratching, consisting of a papulo-vesicular and pustular eruption on and around the sites occupied by the burrows and at some distance from them, mixed with impetiginous lesions, scratch-marks, blood-crusts, or eczematous patches. The abdomen and buttocks are particularly affected, and lesions may also be present on the thighs and arms. In young children the face may be affected, generally with impetiginous lesions, and there is a greater tendency to the production of a widespread eczematous dermatitis than in adults. The feet, too, are more often attacked in children.

**Diagnosis.**—When burrows can be detected and the acarus extracted the diagnosis is no longer in doubt. In cleanly patients, or in those whose occupation necessitates frequent immersion of the hands in water or chemical substances, no burrows may be found, and the diagnosis then rests upon the peculiar distribution and multiformity of the lesions, their discrete character, and a history of infection or of the disease in other members of the family. It should be remembered that scabies is often contracted from sexual intercourse and that *sypilis* may coexist. On the other hand, the induration which sometimes occurs around a burrow on the penis should not be mistaken for that of a chancre. In *pediculosis vestimentis* the upper part of the back and shoulders is the chief site of the eruption, and hæmorrhagic spots will be present. In *urticaria* the essential lesion is a wheal. *Papular urticaria* of children may closely simulate scabies, but no burrows will be found, and the papules and vesicles are preceded by wheals. In *varicella* the vesicles are sparse, discrete, and situated on the trunk, and lesions may generally be found in the mouth.

**Treatment.**—The favourite remedy for scabies is sulphur ointment. The ointment

## SCALP WOUNDS, TREATMENT OF

should be well rubbed into the skin all over the body below the neck after thorough scrubbing with soft soap, particular attention being directed to the parts favoured by the acarus. This should be repeated daily for four or five days, the ointment being left on the body after the bath. If too strong a preparation is used or the treatment is continued for too long, a dermatitis is apt to be set up by the sulphur; a 5-per-cent. ointment is sufficiently strong. Alternative preparations are balsam of Peru 1 dr. to 1 oz., and  $\beta$ -naphthol 15 to 30 gr.; or these drugs may be combined in smaller proportions with prepared chalk and vaselin. Balsam of Peru must be used with care, as it has been known to cause albuminuria. Sulphur baths (potassa sulphurata 1 dr. to the gallon) and powdered sulphur dusted on to the skin and clothing are also useful. The clothes and bedding should be thoroughly disinfected.

S. E. DORE.

**SCALDS** (*see* BURNS AND SCALDS).

### SCALP WOUNDS, TREATMENT OF.

—Scalp wounds may be incised, lacerated, or punctured, and, even when they have sharply cut edges, may be produced by blunt weapons. When the hair is caught in machinery, partial or complete avulsion is liable to occur. The most serious complication is infection, for this process sometimes spreads by perforating veins to the sinuses of the skull, or by lymphatics to the dura and brain. In the treatment of scalp wounds it is essential to ascertain whether there is an associated skull lesion. Palpation by means of a probe will only reveal a depression; a fissured fracture will remain concealed. The lips of the wound should be pulled apart, and it will then be seen whether the instrument which caused the wound penetrated as far as the bone, and whether, if it reached so far, it injured that structure. A fissured fracture has not the serrated appearance of a natural skull suture, and it oozes blood. (For wounds of the skull, *see* HEAD INJURIES.) If the wound is a simple one it will require careful cleansing, the arrest of hæmorrhage, and suture. The surrounding area must invariably be shaved and disinfected. It is best to cut away with knife or scissors any tissue visually contaminated with dirt. Hæmorrhage may be free, because in the dense tissues of the scalp vessels are liable to be half-severed only. If bleeding of this kind is encountered the injured vessel must be cut across completely. Liga-

## SCARLET FEVER

tures are apt to hold badly in the scalp unless the vessels are under-run with a needle. Catgut alone should be used for ligatures in accidental wounds. Usually the suture may be relied upon to arrest hæmorrhage, so that ligation becomes unnecessary. Should cellulitis supervene, the condition of the patient may become very serious (*see* CELLULITIS).

C. A. PANNETT.

**SCAPHOCEPHALY** (*see* SKULL, CONGENITAL ABNORMALITIES IN SHAPE OF).

**SCARLET FEVER** (*syn.* *Scarlatina*).—An acute infectious disease characterized by a cutaneous eruption and faucial angina.

**Etiology.**—Scarlet fever is a disease of temperate climates, being almost unknown in the tropics and seldom seen in subtropical countries. It is rare in the first year of life, especially among breast-fed infants, and becomes more frequent each year, reaching its maximum frequency at the fifth or sixth year. After puberty there is a marked drop in the incidence. The disease seldom occurs in old age. Females are more liable to be attacked than males, but the latter show a higher mortality.

The morbidity and mortality are highest in September, October, and November. Schools play an important part in the spread of the disease, though to a less extent than in the case of measles.

The infectivity is highest in the pre-eruptive and eruptive stages, the contagium being conveyed in discharges from the throat, nose, and ears. Desquamation, formerly regarded as the chief agent in the spread of the disease, is probably innocuous unless contaminated by these discharges.

Most cases cease to be infectious in the course of the second fortnight, unless complicated by discharges from the nose or ears.

The disease is mainly spread by direct contact. Infection usually takes place through the throat, and the virus is likely to gain entrance all the more easily if the tonsils are in an unhealthy state through chronic hypertrophy.

The transmission of the disease by fomites has probably been greatly exaggerated in the past, and is better explained by the existence of "carriers," for after leaving the human body the virus of scarlet fever is short-lived.

Several epidemics have been attributed to infected milk.

## SCARLET FEVER

**Bacteriology.**—The causal agent of scarlet fever has not been discovered, although considerable importance is to be attached to a streptococcus as a secondary invader. Streptococci are almost always present in the throat, in various suppurative lesions, and in the blood of complicated cases. They are absent, however, from the blood at the onset of the disease, even in toxic cases.

**Inoculation.**—Though the micro-organism has not been discovered, scarlet fever is inoculable into man and monkeys. Stickler, in 1899, inoculated ten children with mucus obtained from the mouth or throat of patients who had had a mild attack, and in every case produced typical scarlet fever after an incubation period ranging from twelve to seventy-two hours.

Several experimenters claim to have successfully inoculated monkeys with scarlet fever, but their results are to be received with caution, as these animals are always liable to sudden rises of temperature and transient rashes on the face and neck, and constantly show a branny desquamation.

The most convincing work is that published in 1912 by Landsteiner, Levaditi and Prasek on anthropoid apes. Not only did they produce a typical angina, rash, and desquamation by swabbing the animals' throats with infected mucus, but the histological changes in the skin were identical with those described in human scarlet fever. Their experiments proved that the virus exists in the tonsillar and lingual deposit, the blood, the lymph-glands, and possibly the pericardial fluid.

**Morbid anatomy.**—The only constant change found post mortem is a general hyperplasia of the lymphoid tissue. The liver is usually enlarged and pale. Microscopically the cells show fatty degeneration and, occasionally, areas of focal or central necrosis.

The heart-muscle is pale and soft. Pericarditis and endocarditis are rare. Interstitial myocarditis is more frequent. The renal lesions vary according to the stage of the disease at which death took place. In an early stage congestion only will be found. In more advanced cases parenchymatous or glomerular nephritis is frequent. In some cases interstitial nephritis is present.

Degeneration and hæmorrhagic changes, which may be only visible microscopically, are sometimes found in the suprarenal glands. The pancreas may show interstitial and degenerative changes.

The morbid process in the skin is an acute

hæmorrhagic infiltration situated in the superficial layers of the dermis and epidermis. Mononuclear and polymorphonuclear leucocytes accumulate round the dilated vessels of the dermis and hair-follicles, and invade the epidermis. The epithelium becomes vacuolated and small epidermic vesicles form, enclosing more or less degenerated leucocytes and micro-organisms. The superficial layers become detached and desquamation results. Similar changes take place in the mucous membrane of the tongue, soft palate, tonsils, and pharynx.

**Blood.**—At the onset of the disease there is a moderate leucocytosis, which reaches its maximum intensity during the eruptive period, and gradually declines to normal in convalescence. In complications such as necrotic angina, otitis, and suppurative adenitis the leucocytosis is high. The eosinophils may show a moderate increase at the height of the disease. There is a slight but constant diminution in the red cells and hæmoglobin.

In almost every case up to the fourth day the polymorphonuclears contain one or more of Döhle's "inclusion bodies," i.e. round, oval, or bacillus-like substances, usually situated near the periphery of the cell. Though by no means pathognomonic of scarlet fever, as Döhle claimed, being found in a large proportion of cases of measles, diphtheria, tonsillitis, and other diseases, they are most constant and abundant in scarlet fever, and their absence during the first four days of the disease excludes scarlet fever with a fair degree of certainty.

In the third week, especially in scarlatina anginosa, the serum may yield a positive Wassermann reaction lasting from two to four weeks (Jacobovics).

**Symptomatology.**—In the great majority of cases the incubation period is from two to three days, but it may be as long as seven days or less than twenty-four hours.

The onset is usually sudden. In addition to the symptoms common to all febrile attacks there is a triad, viz. vomiting, headache, and sore throat, which is eminently characteristic of scarlet fever. Vomiting is the most constant symptom, being found in about 80 per cent. of all cases. It may be accompanied by diarrhoea, but constipation is the rule during the febrile period.

Sore throat is usually a prominent symptom, but in mild cases there may be no complaint of it, and in toxic scarlatina it is slight or absent.

## SCARLET FEVER

A sudden steep rise of temperature is the rule, the thermometer registering 103° or 104° F. within a few hours of the onset. The pulse is quicker than in any other acute disease, 140 to 160 pulsations being common even in mild cases. The respiration, on the other hand, is not remarkably affected.

Delirium may be an early symptom, and in young children convulsions may occur at the onset.

**The eruption.**—This may be the first symptom, even preceding by some hours any rise of temperature; but, as a rule, the rash does not appear until about twenty-four hours after the onset. Only in rare cases is it delayed beyond this period. It first appears on the neck and upper part of the chest and then invades the rest of the trunk. It may stop short at the proximal extremities of the limbs, but usually passes down to the hands and feet.

The eruption consists of two distinct elements, an erythema and reddish points. The two are usually combined as a punctate erythema, but either may be present separately on different parts of the body. Thus the palms and soles show only a diffuse or blotchy erythema, and puncta only may be found on the outer aspect of the limbs. The colour of the eruption varies from a brilliant scarlet to a dark red or dusky purple. As a rule, the rash is most pronounced at the flexures of the joints, on the pubes and upper and inner sides of the thighs. The cheeks are brightly flushed, and there is often some turgescence of the skin of the face and eyelids, but the circumoral region shows a striking degree of pallor (Filatow's sign). On the limbs the rash is frequently blotchy and even morbilliform. At the fold of the elbows it is often petechial (Pastia's sign), and the pressure of a band round the upper arm may produce petechiæ at this site (Rumpel-Leede phenomenon).

The rash may last for only a few hours, then disappearing entirely; on the other hand, it may not be developed fully until the second or third day, beginning to fade, after a stationary period of twelve to twenty-four hours, first on the trunk and last on the limbs.

A variable degree of yellowish or pale-brown staining remains; it is most manifest in those cases in which the rash has been most intense.

A certain amount of itching may occur at any stage of the eruption. Miliaria frequently accompanies the specific eruption, its sites of predilection being the neck, anterior folds of

the axillæ, and groins. Urticaria may also be present.

**Tongue and throat.**—The characteristic tongue of scarlet fever is one covered with thick white fur through which project the hyperæmic papillæ, so that the organ resembles a strawberry covered with cream. As the fur peels off, the denuded surface resembles a ripe raspberry.

These appearances are not found in every case of scarlet fever, and are sometimes present in other acute infectious diseases such as diphtheria or measles.

The mucous membrane of the tonsils, faucial pillars, uvula, and palate shows a more or less vivid injection. The colour of the enanthem varies from a slight hyperæmia to an angry red or purple. Follicular deposit is frequently seen on the tonsils.

There is more or less injection of the buccal and gingival mucous membrane. The gums are frequently covered with a thin greyish coating due to catarrhal stomatitis.

**Desquamation.**—The extent and duration of desquamation are closely related to the intensity of the eruption. After a brilliant rash the peeling is profuse and persists for six weeks or more; but after a faint rash, especially in infants, desquamation is slight, lasts only a fortnight or less, and may be absent.

Desquamation usually starts in those areas in which the eruption has first appeared. By the fourth or fifth day a fine powdering may be seen on the cheeks, and by the end of the first week peeling of the neck, the upper part of chest, and the pubes takes place. The process gradually spreads over the rest of the trunk and limbs, the hands and feet being last affected.

Desquamation may sometimes be found on the pubes and adjacent parts, i.e. areas in which the rash is usually most intense, when no trace of peeling can be discovered elsewhere.

The character of the peeling varies according to the area affected. On the face it is little more than a fine powdering; on the trunk, pinholes are produced by shedding of the summits of the miliary vesicles, and, by coalescence with one another, form circinate or polycyclic areas of loosening epidermis.

The most characteristic desquamation is to be found on the palms and soles. The skin of these parts becomes dry and cracked, and the epidermis begins to separate in the subungual region, becoming gradually detached either

## SCARLET FEVER

spontaneously or by the patient in more or less complete strips, which may sometimes form a complete glove or slipper-like cast.

Shedding of the hair and nails rarely takes place.

**Varieties.**—Three principal varieties of scarlet fever are described—(1) simple, (2) septic, (3) toxic.

1. **Simple scarlet fever.**—The great majority of cases belong to this group. The skin and throat manifestations vary in degree. The rash may be bright and general and last for two or three days, or, on the other hand, faint, localized, and transient. It may sometimes be absent (*scarlatina sine eruptione*). The throat is usually more or less injected, there may be some deposit on the tonsils, but any loss of substance is unusual. The general disturbance is not very pronounced, and the temperature, which rarely exceeds 103° F., is usually normal within a week and frequently within three days; in rare cases there is no rise of temperature at all.

2. **Septic scarlet fever** (*scarlatina anginosa*).—This form is characterized by the intensity of the throat manifestations, the severe constitutional disturbance, and the frequency of septic complications. The fauces, palate, and uvula are oedematous and become covered with a yellowish-white exudate. Strings of sticky mucus form on the palate and teeth, the tongue becomes dry and ulcerated, the lips swollen and cracked. There is a profuse acrid nasal discharge which causes redness and excoriation of the nostrils and philtrum. The sub-maxillary and cervical glands become considerably swollen, sometimes forming a collar round the neck. Swallowing is painful and often impossible, regurgitation of fluids through the nose is frequent. As the disease advances, ulceration of the faucial tissues takes place, causing more or less destruction of the tonsils, pillars, uvula, and soft palate. This so-called "necrotic angina" may extend downwards and involve the larynx and even the oesophagus. The rash may be general, intense, and confluent, or ill-marked on the trunk and blotchy on the limbs. The constitutional disturbance is severe. Restlessness and insomnia are the rule; delirium is frequent at night and may occur in the day as well. The temperature is high from the first, and even in favourable cases does not reach normal before the third or fourth week. A blotchy or circinate erythema frequently appears in the second week on the face, elbows, knees, and buttocks.

Complications are much more liable to occur in this than in any of the other varieties of scarlet fever, especially otitis, rhinitis, adenitis, and rheumatism. In fatal cases broncho-pneumonia is usually present. Wasting is a prominent feature in convalescence.

3. **Toxic or malignant scarlet fever.**—This form is by far the rarest. The onset is sudden. There is great prostration from the first, restlessness and delirium are the rule, and there is repeated vomiting. The temperature rapidly rises to 106° or 107° F., but may be subnormal before death. The fauces show some injection, but the throat symptoms, as a rule, are not prominent. Death takes place in 12–36 hours from the onset. In semi-toxic cases death is delayed to the fifth or sixth day.

Cases of *hæmorrhagic scarlet fever* have been described characterized by hæmorrhages into the skin and from the mucous membranes. The writer has not met with such a case apart from purpura arising in convalescence, and is inclined to regard the cases so described as examples of hæmorrhagic smallpox.

**Surgical scarlet fever.**—Scarlet fever is frequently seen after surgical operation and injuries, especially burns. In some of these cases the eruption first appears in the neighbourhood of the wound. It has been suggested that the virus obtains entrance through the breach of surface, but it is more probable that the patients are particularly liable to infection owing to the weakening of their natural powers of resistance by the injury or the circumstances which necessitated the operation.

**Puerperal scarlet fever.**—Scarlet fever may attack puerperal women like other members of the community, but probably many of the cases regarded as scarlet fever are really examples of puerperal septicæmia with a scarlatiniform rash. There is no justification for regarding scarlet fever in puerperal women as especially severe.

**Complications.**—The commonest complications in order of frequency are otitis, albuminuria, nephritis, rheumatism, and tonsillitis. The numerous other complications are much less frequent, occurring in less than 1 per cent. of all cases.

**Otitis.** Inflammation of the external auditory meatus with involvement of the tympanic membrane occasionally takes place. Otitis media, which is far more frequent and serious, may occur during the acute stage or convalescence. It may be caused by the toxins of



## SCARLET FEVER

the disease apart from the throat condition, or be due to the extension of the pharyngeal inflammation by the Eustachian tube, and is therefore most frequent in scarlatina anginosa. It is most commonly found in young children, especially the subjects of adenoids, and is rarely seen above the age of 15.

The *streptococcus pyogenes* and the *staphylococcus pyogenes aureus* and *albus* are the most frequent organisms present. The *pneumococcus* and *pneumobacillus* are sometimes found. Diphtheroid organisms and more rarely true diphtheria bacilli may occur in chronic cases.

In many cases, especially in young children, a discharge from the ear is the first sign of otitis, but it may be preceded by more or less otalgia, with a rise of temperature or exacerbation of pre-existing fever.

The otorrhoea is at first watery, but usually soon becomes purulent. It may last for a few days or persist for months, but in most cases it ceases within a few weeks, and there is little or no impairment of hearing. In a certain number otitis media is complicated by abscesses in or around the mastoid process, shown by œdema and tenderness of the skin behind the ear, which is pushed forward. When the condition is merely due to suppuration of the posterior auricular glands or to periostitis of the mastoid, there is little or no constitutional disturbance. In empyema of the mastoid antrum, which is much less frequent, the œdema is more extensive, the constitutional disturbance is severe, vomiting and rigors occur, the temperature rises to 104° or 105° F., and in default of surgical interference meningitis or cerebral abscess may develop.

Other serious but rare complications of otitis are necrosis of the ossicles, facial paralysis, thrombosis of the lateral sinus, and fatal hæmorrhage from ulceration of the internal carotid.

The internal ear may be affected by extension of the inflammation from the middle ear, or may be involved independently. Statistics show that from 1.5 to 27.5 per cent. of cases of acquired deaf-mutism are due to scarlet fever (Macleod Yearsley).

**Adenitis.**—A general enlargement of the lymphatic glands takes place during the acute stage, the submaxillary and cervical being especially liable to be affected. The inflammation is usually of moderate degree, and complete resolution takes place, but early cervical adenitis may end in suppuration or chronic hyperplasia. In very severe cases cervical

adenitis may form the starting-point of a diffuse cellulitis which is almost invariably fatal. At a later stage, usually towards the end of the third week, a recrudescence of the cervical and submaxillary adenitis may occur, often in a more marked degree than during the eruptive period. This late adenitis is usually attended with a steep rise of temperature and often accompanies the onset of nephritis.

Like otitis, this complication rarely occurs after puberty. It is most apt to follow severe attacks, especially if oral sepsis be present. Complete resolution is the rule, but suppuration occurs in about one-third of the cases.

**Rheumatism.**—This is one of the earliest complications. Its time of predilection is towards the end of the first week or the beginning of the second. It is more frequently met with in older children and adults, especially those who have had a previous attack of rheumatism, than in younger persons, and in females than in males. It is commoner after severe than after mild attacks, and it is therefore not surprising to find that its frequency has declined of late years. The smaller joints are chiefly affected, especially the hands. It is usually of short duration, and readily yields to treatment. Although cardiac complications rarely arise in fever hospitals, Poynton has shown that cases which have suffered from scarlatinal rheumatism in hospital frequently develop within a month of their discharge other symptoms of rheumatism, especially chorea and signs of organic heart disease. Complete resolution of the arthritis is the rule. Suppuration only occurs in septic cases, usually in association with other signs of pyæmia.

**Nephritis.**—No other acute disease is so often complicated by nephritis as scarlet fever. The frequency of the complication varies in different epidemics. Like most of the other complications, it has tended to become less frequent in recent years. Its frequency bears no relation to the character of the diet, its incidence being practically identical in patients who have been fed on milk only and in those who have been given a meat diet (Pospischill and Weiss). A familial disposition to scarlatinal nephritis is frequently observed.

The onset is usually in the third week. The state of the urine may be the only indication of the condition, but there is usually more or less constitutional disturbance with headache, nausea, vomiting, and pyrexia. Pain in the back is exceptional. The face is pale and puffy, but it is unusual to find any great

## SCARLET FEVER

degree of anasarca or dropsy except when the initial attack of scarlet fever has been overlooked. In such cases pulmonary or glottic œdema may occur and prove fatal.

The urine is diminished in quantity, and varies in colour from a smoky red to a port-wine tint. The nitrogenous output is small, and there is also a great decrease in the excretion of chlorides and phosphates. The microscope shows the presence of red cells and hyalin, fatty and blood casts. Uræmia is exceptional. It is usually heralded by drowsiness, repeated vomiting, slight twitching of the limbs, and a considerable diminution of urine, but occasionally a generalized convulsion is the first sign. Amaurosis may be present, and transient paralyses may occur, especially hemiplegia, with or without aphasia. The symptoms, though severe, usually yield to treatment, and death from scarlatina uræmia is exceptional. The great majority of nephritis patients recover after an illness of one to seven weeks. In a few cases the nephritis becomes chronic, though death may be postponed for fifteen years or more.

Some cardiac disturbance is often associated with nephritis. Dilatation of the heart, gallop-rhythm, and enlargement of the liver may occur, the clinical picture more closely resembling that of cardiac than that of renal disease.

Nephritis is usually distinguished from albuminuria by the presence of blood in the urine, but probably many cases returned in statistics as albuminuria, especially those occurring in the third week in association with adenitis, are really examples of non-hæmorrhagic nephritis.

During the acute stage transient albuminuria is very frequent, and usually disappears before the temperature becomes normal. Orthostatic albuminuria is not infrequent in convalescence.

**Circulatory system.**—After an initial rise the blood-pressure falls during the early weeks of the disease in about a quarter of the cases, the extent and duration of the depression being related to the character of the attack. The normal tension is usually re-established by the fourth week. In only a small proportion of the nephritis cases is the blood-pressure raised, and, except in uræmia, the hypertension is not excessive.

Heart murmurs are frequent during the febrile period, but are usually due to slight dilatation and not to valvular disease. Endocarditis is much less frequent than was formerly

supposed. It may be an early phenomenon associated with scarlatinal rheumatism, or occur at a later period independently. It is rare for it to be fatal or to clear up entirely; as a rule it becomes chronic. Pericarditis is also rare. Like endocarditis, with which it is often associated, it may accompany rheumatism or nephritis, or occur independently. It is usually dry or sero-fibrinous, but it may be purulent, especially when it is secondary to pleurisy.

Myocarditis is more frequent than either endocarditis or pericarditis. It is manifested by cyanosis, dyspnoea, fainting attacks, and other signs of cardiac failure, or be the cause of sudden death.

Functional disturbance of the myocardium without definite anatomical lesions is common, and is manifested by tachycardia, bradycardia, and various forms of arrhythmia.

**Respiratory system.**—The larynx is rarely affected. A mild degree of catarrhal laryngitis is sometimes present during the eruptive period. Far more important is the ulcerative laryngitis which complicates septic cases and results in laryngeal perichondritis and necrosis of the cartilages. Progressive dyspnoea results, and death usually takes place from bronchopneumonia.

In striking contrast with measles, pulmonary complications in scarlet fever are uncommon; bronchitis, broncho-pneumonia, lobar pneumonia, pleurisy, and empyema occurring each with a frequency amounting to less than 1 per cent.

**Alimentary system.**—Ulcerative stomatitis occurred in 1.02 per cent. of the Metropolitan Asylums Board's cases. It is usually the appanage of septic cases, but may follow a mild attack. Secondary tonsillitis was found in 1.80 per cent. It is commoner in adults and in older children than in early life. It may be associated with cervical adenitis and nephritis in the third or fourth week, or may occur independently later. Vincent's angina may appear in convalescence either alone or associated with ulcerative stomatitis. Retropharyngeal abscess may be secondary to septic angina or to suppurative cervical adenitis. Catarrhal gastritis and enteritis are not infrequent, especially in young children during the acute stage. Slight and transient enlargement of the liver is common in the acute stage. Jaundice is rare. It is usually catarrhal and rapidly subsides, but a few cases of icterus gravis have been reported.

Streptococcic peritonitis, which is a very

## SCARLET FEVER

rare complication, may be associated with nephritis or develop independently.

**Nervous system.**—Meningism and serous meningitis may occur at the onset, especially in severe attacks. Purulent meningitis is usually the result of otitis, less frequently of rhinitis, but may arise independently as a pyæmic manifestation. It is usually due to streptococci, rarely to staphylococci. Cerebral and cerebellar abscess may also follow otitis. Hemiplegia, though uncommon, is the most frequent form of paralysis in scarlet fever. It is usually due to embolism following endocarditis. A few cases of peripheral neuritis have been recorded. Epilepsy following scarlet fever is posthemiplegic, or occurs independently in the predisposed. Chorea is an occasional sequel. Various psychoses, such as manic-depressive insanity and dementia præcox, may occur during convalescence.

**Rhinitis.**—Purulent rhinitis is constant in septic cases, and serous or purulent rhinorrhœa is frequent even in mild attacks when the patients are subjects of adenoids. The discharge may be very persistent owing to infection of the accessory nasal sinuses. The lachrymal duct may be involved and dacryocystitis result. The causal organism is usually the streptococcus, but diphtheritic rhinitis is not infrequent in convalescence.

**Skin.**—Herpes facialis was found in 6.5 per cent. of my 413 cases in the acute stage. Herpes zoster is exceptional. Boils and abscesses are frequent in convalescence. Every variety of purpura may occur. More than a third of the reported cases of purpura fulminans have followed scarlet fever. Striæ atrophicæ on the back and knees are not uncommon after severe attacks. Gangrene of the skin may follow cellulitis of the neck, or more rarely occur in disseminated foci on the trunk and limbs. Pemphigus and dermatitis herpetiformis are occasional sequelæ.

**Association with other diseases.**—Scarlet fever may be associated with any of the other acute infections, but has a predilection for diphtheria. Post-scarlatinal diphtheria, which was once attended with a very heavy mortality, is now, thanks to antitoxin, usually a mild disease.

**Relapses.**—Although many so-called relapses are really primary attacks, the initial illness having been wrongly diagnosed, relapses do undoubtedly occur in about 1 per cent. of all cases. They are most likely to develop between the third and fifth weeks, and are oftener seen in children than in adults. The course

of the relapse is usually milder than that of the primary attack. Relapses must be distinguished from erythemata accompanying other streptococcal manifestations, such as tonsillitis or nephritis, from other eruptive fevers, and from drug rashes.

A second attack of scarlet fever some months or years after the first is still more uncommon than a relapse, but genuine examples are occasionally seen in fever hospitals where the nature of each attack can be verified.

**"Return cases."**—Discharge of a patient from hospital after apparent cessation of infection may be followed by one or more cases breaking out in the household to which he has returned. These so-called return cases are not peculiar to fever hospitals, but may also occur in private practice, when the patient has been released from isolation and been allowed to mix with other members of the family. The frequency of return cases in fever hospitals is usually from 3 to 6 per cent.

The residence in hospital of the primary or infecting case is usually two to three days longer than the average, mainly owing to the fact that a large proportion of these patients have had rhinorrhœa, less frequently otorrhœa, or both rhinorrhœa and otorrhœa, during their illness, and had been detained until these discharges had ceased. Patients discharged before desquamation is complete do not show a higher return-case frequency than those who have been isolated until they have finished peeling. There is no relation between the character of the attack in the primary case and in the return case. Severe attacks, however, are unusually frequent among return cases. Among 305 return cases the death-rate was 7 per cent., as compared with a mortality of 3.7 per cent. among a total of 8,309 scarlet fever patients (Sørensen).

The occurrence of return cases cannot be fully explained or prevented until the scarlet fever virus is identified. Hitherto the various preventive methods adopted have been quite ineffective to reduce their frequency.

**Diagnosis.**—The diseases most frequently mistaken for scarlet fever are measles, rubella (q.v.), varicella, tonsillitis, and erythemata due to various causes.

The prodromal scarlatiniform rash most often leads to the mistake in diagnosis from *measles*; this is distinguished by the coexistence of Koplik's spots and catarrhal signs, and the absence of the characteristic throat and tongue phenomena of scarlet fever.

## SCARLET FEVER

A prodromal scarlatiform rash may also occur in *varicella*, but is usually transient, and, like the prodromal rash of measles, is not followed by desquamation. Acute *tonsillitis* is often mistaken for scarlet fever, not only on account of the throat appearances, but also from the occasional presence of an erythema. This, however, is non-punctate, and is usually localized and transient. There is no subsequent peeling of the tongue or skin.

Other *erythemata* which simulate scarlet fever include drug rashes, especially those caused by quinine and belladonna, enema rashes, septic rashes such as occur after an operation or in the puerperium, and erythemata due to alimentary disturbance or of unknown causation. None of these eruptions, however, is accompanied by the bucco-pharyngeal symptoms of scarlet fever, nor does the subsequent desquamation, if present, resemble that of scarlet fever. Erythema scarlatioides is distinguished by the absence of sore throat, by its tendency to relapses and recurrences, and by the shedding of the hair and nails as well as of the skin.

**Prognosis.**—Apart from toxic and septic cases the prognosis of scarlet fever is extremely good. During the last forty years there has been a steady decline in the case-mortality from scarlet fever in the Asylums Board's hospitals. The death-rate in these hospitals is always higher among males than among females for every age group.

Age is an important factor in the prognosis. The mortality is highest in the first year of life, and gradually declines in subsequent years.

Complications, as a rule, are commoner and more severe in younger than in older patients. Apart from toxic cases in which the throat symptoms are not prominent, the prognosis depends to a great extent on the severity of the angina. Of the commoner complications the most serious, as much on account of their remote as of their immediate effects, are otitis and nephritis; of the rarer complications, ulcerative laryngitis and suppurative meningitis are the gravest, and almost invariably betoken a fatal issue.

**Treatment.**—The patient should be kept in bed in a room with a temperature of about 60° F. During the febrile period milk diet is advisable, but when the tongue cleans and the temperature falls, bread-and-butter and milk puddings may be allowed, and in another few days the patient may resume his ordinary diet. A blanket bath should be given daily, and the

bowels should be moved at least every other day. It is advisable to keep every case in bed until the twenty-third day to avoid the risk of nephritis and a tendency to cardiac dilatation. The urine should be tested every other day for albumin. The fauces should be syringed or irrigated with a warm solution such as the following :—

R $\bar{y}$  Pot. chlor. gr. x.  
Tr. myrrhæ ℥x.  
Glycer. borac. ʒi.  
Aq. ad ʒi.

Sore throat may be relieved by fomentations to the neck and by giving small pieces of ice to suck. Restlessness, insomnia, and delirium are often benefited by tepid sponging. The most useful drugs in such conditions are chloralamide and trional. Hyperpyrexia should be treated by cold or tepid sponging; antipyretics should be avoided. The value of alcohol in scarlet fever, as in other acute infections, has been greatly exaggerated. For the prostration and asthenia associated with hypotension occurring in severe attacks, adrenalin chloride 1 in 1,000, in 5-minim doses, should be exhibited.

Though occasionally good results have followed the use of a polyvalent antistreptococcal serum, either alone or in combination with a streptococcal vaccine, the consensus of opinion in this country is that the serum and vaccine treatment of scarlet fever is unsatisfactory.

Salvarsan and neosalvarsan have recently been accredited with a decidedly antipyretic effect and rapid disappearance of the necrotic tissue in septic cases. Encouraging results have also been reported from the use of the serum of convalescents.

In *otitis* early paracentesis is usually recommended by aural surgeons, but is seldom carried out, as otorrhœa is usually the first indication of the condition. When discharge occurs the ear should be syringed every four hours with a warm boric solution, for which, if the discharge persist, a solution of biniodide of mercury (1 in 5,000) or hydrogen peroxide (1 in 1,000) should be substituted. On the appearance of a mastoid swelling an incision should be made down to the periosteum, and any carious bone removed. A radical operation may be required later.

**Nephritis.**—The diet should be restricted to milk, lemonade, and barley water. Meat extracts should be forbidden. The patients should lie between blankets, and the bowels

## SCHISTOSOMIASIS

should be moved freely every day. As the albumin diminishes, the diet may be increased, but it should be as salt-free as possible.

In uræmia, venesection is valuable. Pilocarpine should be avoided. Chloroform is required if the convulsions are subintractant. The cardiac dilatation associated with nephritis is benefited by the use of digitalin internally and subcutaneous injections of caffeine or camphor.

In *rheumatism* the affected joints should be wrapped in cotton-wool, and aceto-salicylic acid should be given in large doses. Pyæmic arthritis requires early incision.

J. D. ROLLESTON.

### SCHICK'S TEST (see SEROLOGICAL DIAGNOSIS).

**SCHISTOSOMIASIS.**—Under the generic title of *Schistosoma* are included three different species of trematodes, popularly known as the *Bilharzia* worms, which infest the human body. The more widely distributed of these varieties are: (1) *Schistosoma hæmatobium* (*Bilharzia hæmatobia*), with its marked predilection for the urinary tract; and (2) *Schistosoma* (or *Bilharzia*) *mansoni*, with an even more selective attraction towards the intestine and its associated viscera. Another variety, of much more localized geographical distribution, is (3) *Schistosoma japonicum* (*Bilharzia japonica*).

Man becomes infested with schistosoma, whatever the species, by way of the skin or mucous membranes, from water containing active cercariæ, the larvæ of the developing trematode. Evidence of the actual moment of infection may sometimes be forthcoming as a pricking sensation or itching of the skin, after bathing in an infected pool. Then succeeds a stage of incubation, lasting from four to six weeks, with a complete absence of symptoms. At the end of this time the cercariæ have become transformed into mature worms, of both sexes, which are found lying in large numbers in the main venous channels of the portal system. While undergoing this metamorphosis they secrete various toxins which produce a series of invasion symptoms of very definite character, including an irregular fever, diffuse urticaria, enlargement of the liver, and signs of pulmonary and intestinal irritation. These symptoms last for 10-30 days, are associated with a marked eosinophilia, and manifest themselves in five to eight weeks after the original entry of the cercariæ.

A second incubation period of four to eight weeks follows, during which the coupled worms set forth on their travels throughout the portal venous system and the females are busy depositing ova. These activities result in the formation of definite pathological lesions and, towards the end of this second quiescent stage, in the discharge, from the now completely infested human body, of ova, fully fertilized, and each containing a living embryo, into the urine and fæces. Should these ova gain access to water while the contained embryo, or *miracidium*, is still living, the chitinous shell bursts, and the miracidium finds itself free and swims off in search of certain fresh-water snails, by which it is strongly attracted, and in the livers of which the cercariæ are formed. In about six weeks, when the cercariæ are fully mature, they are ejected into the surrounding water and at once become possible sources of infection, and may penetrate any human skin or mucous membrane with which they come into contact.

In a country in which the disease is endemic, the geographical peculiarities, the conditions of life, and the social habits and insanitary customs combine to render infection and re-infection almost inevitable, so that the incidence of schistosomiasis must always be very high. A single infection may be recovered from in 15-20 years; but, as the pathological changes become more and more severe, and septic and even malignant complications almost naturally follow, no hope of any but temporary relief can be expected in reinfected cases in endemic areas. Cure can only be assured when all the worms are dead and all the ova have either been discharged from the body or are securely encapsuled in fibrous tissue.

**The Fairley reaction.**—The existence of mature worms in the body may be determined by this reaction, in which livers of infested snails are used as antigen; it is of value not only as a means of diagnosis, but as a criterion of the efficiency of treatment, especially when applied by the intravenous route. In new infestations also, both individually and on a large scale, the reaction is especially helpful in making a definite diagnosis before ova are present in the urine or dejecta.

### URINARY SCHISTOSOMIASIS

**Etiology.**—The infestation is by *Schistosoma hæmatobium*, which is distributed widely all round the North and East Coasts of Africa, attaining its maximum intensity in the Egypt-

## SCHISTOSOMIASIS

tian Delta and along the Nile Valley; and, to a less degree, in South Africa. Its distribution extends inland along river courses even to the Orange Free State and the Transvaal, all through Central Africa, and out on to the West Coast. Outlying centres such as Cyprus, Arabia, and Mesopotamia are also now endemically infested, but here, as elsewhere, the original source of infection is almost certainly Africa.

Its persistence depends upon the presence, in favourable pools or watercourses, of certain fresh-water snails of the genera *Bullinus* and *Physopsis*, the particular hosts in which the cycle of its life-history is completed. Naturally, the agricultural labourer will be infested most of all; and in Egypt evidence is forthcoming that a painless hæmaturia, the essential sign of this disease, has existed throughout the country from time immemorial. The ova of the worm have been discovered in urinary calculi from mummies of the dynastic era.

*S. hæmatobium* (Fig. 87) is a bisexual trematode. The male is 11–15 mm. in length and 1 mm.

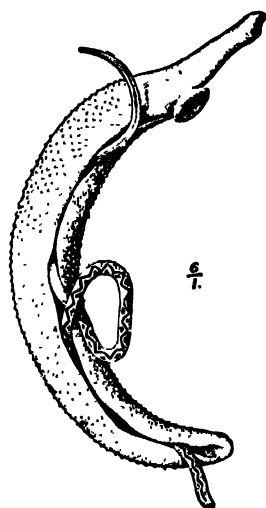


Fig. 87.—*Schistosoma hæmatobium*, male and female. (After Looss and Manson-Bahr.)

in breadth, grey or white in colour, and tapering to each end. It carries an anterior and a ventral sucker, and is covered with cuticular prominences. The worm is really flat like a leaf, its margins being folded towards the ventral surface to form a canal, in which part of the female lies during fecundation. The female is cylindrical, much longer and more filiform than the male, 2 cm. long by 0.25 mm. broad, and tapering. The surface is smooth; and, when coupled, the central part lies within the

gynecophoric canal of the male, but both anterior and posterior portions remain free. The uterus contains many *terminal-spined ova*. These ova are rounded spindle-shaped, 0.16 mm. in length by 0.06 mm. in breadth, light yellow in colour, bearing a short stout spine at their posterior end, and containing a living embryo, or miracidium, inside a definite membrane.

The coupled worms, lying in the portal venous system, eventually travel against the blood-stream to the furthestmost possible limit allowed by the diameter of the vessel. The female then leaves her

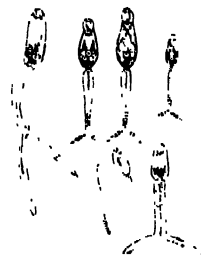


Fig. 88.—Fork-tailed cercariæ.  $\times 25$ .

partner and advances until she completely blocks a venule. The ova are now deposited with the spines pointing in the direction of the blood current. After withdrawal of the worm, the blood-stream forces them through the vessel-walls and they appear shortly afterwards in the urine (Fairley and Manson-Bahr).

When the ova are voided and reach water the shells burst, and the freed embryos swim to and enter their special snails, *Bullinus dybowskii*, *B. innesi*, or, in South Africa, *Physopsis africana*. The cercariæ into which they are transformed (Fig. 88) can survive 24–36 hours after emission from the snail, in the presence of oxygen and warmth. They attach themselves to the skin of their victim by means of their suckers and find their way into the subcutaneous tissues, where the tail is lost and they soon reach the liver, to become mature worms, either male or female. In six weeks' time the now fully-developed worms are lying in numbers in the venous spaces of the liver and the portal venous system, ready for their journey to their ultimate destination, the portal, inferior mesenteric, and hæmorrhoidal veins, the vesico-prostatic plexus and, occasionally, the vena cava and pulmonary vessels.

**Pathology.**—The effects of this infestation are produced by toxins secreted by the worms and also by irritation of the tissues by the ova deposited so abundantly within them. The toxic symptoms of the earlier invasion-stage have been noted. Once the disease has been thoroughly established, the ova, deposited in the submucous tissues and streaming through the mucous membranes, excite much epithe-

## SCHISTOSOMIASIS

lial proliferation on the surface and an inflammatory round-celled infiltration, containing many eosinophils, beneath them or in subcutaneous tissues.

In urinary schistosomiasis minute papules, with a ring of congestion around them, are first formed on the posterior wall of the bladder; then follows a more or less general infiltration of the deeper layers of the mucous membrane, usually in patches. So intense may be the process that a yellowish-brown deposit of calcified ova may entirely replace the normal mucous membrane, while the deep infiltration is so marked that the bladder becomes a much thickened non-elastic fibrous bag, and very prone to secondary septic infection. Papillomata, varying in size and type, also form and may assume large proportions. These and other sessile masses of similar structure may become sarcomatous, or less often carcinomatous, with all the associated dangers locally and after dissemination. Somewhat similar infiltrative changes occur in the ureters, with loss of elasticity and dilatation, thus contributing to the virulent septic infection which is the usual end in severely infested patients. The kidney shares largely in this septic process, though actual bilharzial changes in it are not common. The prostate, the vesiculæ seminales, and the urethra and periurethral tissues are all frequently involved in the infestation, and cirrhotic changes may occur in the liver.

**Symptomatology.**—The symptoms of skin irritation and of toxic invasion are followed, after 4–8 weeks or more, by a burning sensation towards the end of micturition, an increased urgency, and ultimately a definite terminal hæmaturia, which in an infested country at once calls for microscopical examination and the search for the characteristic terminal-spined ova. In most cases no evidence of the infection is forthcoming, and the first sign may be a painless hæmaturia which may persist almost indefinitely without much more trouble.

The hæmaturia varies considerably in quantity and, since it may be associated with severe toxic effects, may produce much general anæmia and weakness. In almost all cases there is some irritability of the bladder and pain and tenderness, varying in intensity and situation, about the pubes, perineum, neck of the bladder, penis, or loins. A tender prostate, an increase in pain and in bleeding on exertion, or after the insertion of a sound, with or without the discovery of a small uric-acid or oxalate stone, all point towards the diagnosis, which

is made absolute by the discovery of the ova. This usually presents no difficulty if the last portion of the urine is taken for examination and the deposit centrifugalized and examined at once on a slide. Rarely, hæmospermia may be quite an early sign and indicate involvement of vesiculæ and prostate. Later, the case becomes one of chronic cystitis with hæmaturia. The symptoms progress, *pari passu* with the advancing pathological changes, through all the stages of a virulent septic infection of the whole urinary tract, complicated, it may be, by malignant disease, both local and general.

Vesical calculi are very often found; the first are composed of oxalates and uric acid, which salts become deposited on a mass of ova entangled in mucus, a piece of papilloma, or on a bloodclot; later, when the urine becomes alkaline, phosphates form, either on existing stones or as concretions on abraded bilharzial lesions, in any part of the urinary tract.

The male urethra is sometimes severely affected. Infiltrated patches occur in the mucous membrane, with much round-celled deposit in the periurethral tissues. The usual result is perineal fistulæ, which may cause great destruction of the urethral canal, the portion most affected being that between the posterior margin of the scrotum and the triangular ligament. A similar condition in the penile urethra leads to multiple small fistulæ and marked lymphatic œdema of the glans penis and of the subcutaneous tissues of its body. Considerable false elephantiasis of penis and scrotum may develop.

Urinary schistosomiasis in women produces extensive changes and destruction in the bladder and ureters; vesico-vaginal fistulæ and not infrequently large phosphatic calculi may form. Papillomata appear at the vulval orifice, in the vagina, or on the cervix uteri, and all the various infiltration changes occur in the vaginal and urethral mucous membranes.

Rectal symptoms, with passage of blood and mucus containing terminal-spined ova, may be produced without any evidence of *Schistosoma mansoni* infestation; but in endemic areas the association of the two varieties of infestation must be frequent. Only very rarely is hæmoptysis traceable to bilharzia.

**Diagnosis.**—In endemic areas the diagnosis is generally obvious; and even if the symptoms are not always convincing, confirmation is easily obtained by the microscope. A cysto-

## SCHISTOSOMIASIS

scopic examination may be helpful and reveal minute papules about the trigone and ureteric openings, patches of hyperæmia and, later, tiny glistening globules of calcified ova clustered upon an infiltrated patch of mucous membrane. Papillomata may also be seen and yellowish wet-sea-sand areas of more intense infiltration, of varying size and extent. This sandy change may later involve almost the whole field. All the various structural and septic changes in ureters and bladder may be found, but later may be obscured by turbid urine in advanced cases of the disease.

The urine should always be examined carefully, and living ova may be hatched out by adding warm water. Many of the ova are dark and calcified. As the disease progresses, the urine contains pus, blood, débris, oxalate crystals and, later, phosphatic masses and concretions. Before long all the characters of an intensely alkaline urine are present, and it becomes watery and takes on a peculiar greenish-grey tint. The characteristic terminal-spined ova may also be found in the fæces, especially in the mucus round it, and rarely in the sputum.

The blood picture is that of an intense eosinophilic leucocytosis, the same features predominating in the microscopical examination of sections of the various pathological lesions.

**Treatment.**—The treatment of schistosomiasis of all forms consists first in a *general prophylaxis*, which includes the protection of the water supply from fæcal or urinary contamination, and the destruction of the intermediary hosts, the fresh-water snails—both of them very difficult problems in actual practice; and then in *personal prophylaxis*, which, in an infested country, may prove equally uncertain.

*Symptomatic* treatment follows the usual lines adopted for irritation and inflammation of the urinary tract, and the recognized régime of diet and drugs, both local and general, must vigorously be adopted. Relief sometimes follows the use of extract of male fern in 10-min. doses, in capsule, three times a day, with a buchu and hyoseyamus mixture. Other alkaline diuretics, the benzoates, methylene-blue, and urotropine have their value, and acid sodium phosphate frequently relieves the irritation caused by the early alkalinity of the urine. Various infusions of local “herbs and simples,” and especially bizr-el-killah, shaw-wash-el-durra, and native barley, are employed,

often with great relief, as well as large quantities of water, barley water, and diuretic mineral waters. A tonic treatment, particularly with iron, is also needed; and, when it can be borne, copious irrigations of the bladder with dilute solutions of permanganate of potash, eusol, oxygen water, and even silver nitrate, have a limited usefulness. Stones should be removed by lithotripsy; at a late stage, when sepsis has claimed the victim for its own, extensive perineal drainage of the bladder must be performed and affords temporary relief.

Urethral fistulæ must be dealt with radically on proper surgical principles, and decortication of a much swollen elephantiac penis, with subsequent grafting, is often called for. A malignant change in the glans penis or in the bladder may require very extensive operation; but at best this is purely palliative, like the operations required for the advanced disease in women. Deposits of bilharzial tissue in skin, subcutaneous tissues, the erectile tissue of penis, spermatic cord, and elsewhere may sometimes be completely excised.

The *specific* treatment by the intravenous injection of tartar emetic, as first adopted by Christopherson, has often given extraordinarily good results and must be followed in all but the most advanced cases. Day has elaborated the treatment into a system which is now the routine method adopted at Kasr-el-Ainy Hospital, Cairo, and is being applied to upwards of 200 new patients a month. Only cases with active disease, with living ova either in urine or in fæces for good effects are obtained in intestinal schistosomiasis also are treated; the contraindications are advanced cardiac, arterial, or renal disease. A freshly prepared 6-per-cent. solution of tartar emetic in sterilized water is used, the initial dose being 1 c.c. (or 1 gr.) of the drug. The injection is made with the usual precautions into the median basilic vein, and the greatest care taken to avoid leaking of the solution into the subcutaneous tissues beside the vein. The syringe is charged with the dose, the needle, which must be very sharp, is dipped into boiling water to clear it of all trace of solution, and the vein cleanly punctured. No injection is made until a quantity of blood flows back into the barrel of the syringe and there is no trace of swelling in the skin or about the vein. After the injection, which is done within two to three hours after the morning meal, the patient lies down for one to two



## SCHISTOSOMIASIS

hours. Three courses of treatment are given. The first begins with a dose of 1 gr., followed by 2-gr. doses every second day until five injections have been given—9 gr. in all. Then follows an interval of seven days. The second course starts with a 2-gr. dose and is of the same length as the first, 10 gr. of the drug being given, or even more—2½ gr. for a dose—if no bad effects are noted. After another interval of seven days a third course, precisely similar to the second, is given. A fourth may follow if ova are still present or a recurrence ensues. The quantity of tartar emetic in a full treatment is thus usually 29–30 gr.

The urine is examined macroscopically at each injection and microscopically after each course. The hæmaturia generally ceases by the end of the first week, and the number of ova is much diminished. By the end of the second week most of the ova fail to hatch out, and gradually all the miracidia perish, till by the end of the third week they are all dead, and masses of black ova held together by mucus may be passed.

The same changes occur in intestinal cases in the same periods of time. Day concludes that the effect of the antimony is to stop the active egg-laying; and, though a moderate dose will destroy the ova already deposited, a full and prolonged course or series of cumulative courses is required to kill the parent worms. Children 8–15 years of age have for their first course ½ gr., then ¾ gr., then 1 gr., which may even be increased to 1½ gr. The doses for the second and third courses are 1–1½ gr. A woman rarely takes more than 1½ gr. for a maximum dose.

The bad effects which sometimes occur are slight giddiness and cough, perhaps vertigo and headache and, rarely, vomiting. If these symptoms last for more than a few hours the next dose must be reduced till tolerance is gained. Locally, an inflammation, or even an abscess, may occur at the site of injection, in which case emetine must be substituted for the tartar emetic; a 3-per-cent. solution in sterilized water is used, 1 gr., increased to 1½ or even 2 gr., being given exactly in the same way as the antimony and continued till all signs of inflammation have subsided. The emetine keeps up a mild action and saves valuable time till the more potent drug can be resumed.

In urinary cases with cystitis or other inflammatory condition, not directly due to bilharzia, surgical treatment must be adopted

after the course of antimony; and cases of perineal fistula with extensive subcutaneous infiltration should be submitted to this course before operation.

Colloid antimony and its ammonium salts and acetyl-p-aminophenyl stibiate of soda are also on trial.

### INTESTINAL SCHISTOSOMIASIS

**Etiology.**—*Schistosoma mansoni* very closely resembles *S. hæmatobium*, but both male and female are rather smaller, and are especially found in the lower mesenteric veins, particularly around the rectum, and in the branches of the portal vein within the liver. The eggs are oval, with a stout, well-marked lateral spine, and are slightly shorter and broader than those of *S. hæmatobium*. They are found particularly in the coats of the intestinal wall, and pass thence to be voided with the fæces, often in the thick mucus enveloping the solid dejecta. Their miracidia, after hatching out, enter a *Planorbis* snail—*P. boissyi* in Africa, *P. olivaceus* and *P. centrimetralis* in South America.

*S. mansoni* occurs mainly in Egypt and the Sudan, and sporadically but not endemically in other parts of Africa. It is here frequently associated with *S. hæmatobium*, but in the New World, where it has an extensive distribution in Venezuela, the West Indies, and Central America, it exists alone.

**Pathology.**—The lesions are chiefly confined to the large bowel, and are at their worst in the sigmoid and the rectum. They consist mainly of infiltration of the mucous membrane and formation of papillomata, some of which may subsequently slough off and leave characteristic ulcers. In places there may be a very intense sowing of the interior of the gut with numerous papillomata, associated with great thickening and hypertrophy of its walls and a solid diffuse round-celled and eosinophilic infiltration of the mesocolon or mesorectum. The whole rectal mucous membrane may be diffusely infiltrated and protruded from the anus, where also large pendulous papillomata, with much redundant infiltrated mucous membrane, may be present. Extensive fistulæ, similar in origin to the urethral fistulæ of *S. hæmatobium*, may also form and tunnel the surrounding area for a considerable distance. A peculiar pipe-stem cirrhosis of the liver is frequently met with, and deposits of ova, with surrounding nodules and infiltrations, occur in the mesentery and mesenteric glands, in the spleen and

## SCHISTOSOMIASIS

pancreas, and scattered throughout the visceral and parietal peritoneum.

**Symptomatology.**—The symptoms of intestinal schistosomiasis are very variable. The condition may exist for a long time without any definite signs, the discovery of the specific ova in the faeces first establishing the diagnosis. The toxic symptoms and the eosinophilia are much the same as with *S. hæmatobium*, and may be followed after the proper interval by a chronic afebrile diarrhoea, with the passage of blood and mucus, easily ascribable to dysentery. When the disease is in the rectum, troublesome tenesmus, prolapse of infiltrated mucous membrane or of papillomata on defæcation, the passage of a thick, glairy, blood-stained mucus, and ultimately a permanent prolapse, are characteristic, and ova are readily found; while still later the fistulae around the anus are obvious. Either accompanied by dysenteric symptoms, or often quite apart from them, large hard movable tumours may be felt at any point in the course of the large intestine, especially about the caecum, the transverse colon, and the sigmoid; these form a striking feature of the disease in Egypt. The general condition in all the various types of the disease deteriorates very considerably, and anæmia, emaciation, and weakness are extreme.

The **diagnosis** is at once confirmed by finding the lateral-spined ova; their discovery distinguishes the disease from bacillary or amœbic dysentery, with which, however, they may be associated. Sigmoidoscopy will often detect early infiltrations or papules, and should always be employed. Papillomata presenting at the anus may be removed and examined, and generally show ova, as does also a smear taken from the surface of the infiltrated mucous membrane.

The general **prognosis** in endemic areas and the hope of cure are dependent upon conditions similar to those in urinary schistosomiasis. Advanced cases are equally uncertain of permanent relief, if not, indeed, hopeless. It is possible to cure a single infection after removal from risk of infection.

The **treatment** of intestinal schistosomiasis is largely prophylactic, on similar lines to those described above. Symptomatically, it is that of dysentery without the drugs specific for that disease.

The treatment by intravenous injections of tartar emetic, as already described, must be rigidly followed in the active stages of the

disease, and very considerable benefit, if not cure, may result. Locally, much relief follows the removal of rectal papillomata, or of a prolapsed ring of infiltrated mucous membrane, and radical operations on the broadest lines for the fistulae all round the anus must be carried out. Early cases of prolapse do well after Lockhart Mummery's operation by a deep semicircular incision between the rectum and the coccyx. Finally, a laparotomy, followed by an incision into the thickened gut and its subsequent suture, is sometimes very successful in the case of large bilharzial tumours of the colon, affording relief from the abdominal pain with apparent disappearance of the tumour and cessation of the dysenteric symptoms. Very rarely epithelioma develops around the anus and requires radical removal; the massive epithelial growths sometimes found in this situation must be similarly treated.

This particular infestation is seen at its worst in Egypt; elsewhere it produces comparatively mild symptoms.

### SCHISTOSOMIASIS OF THE FAR EAST

This infestation is almost confined to China and Japan and the Philippines, and closely follows the distribution of its special intermediary snail, *Blanfordia nosophora*. It is very common in all rice-growing districts in those countries, the incidence, especially as regards sex, varying with local customs in particular districts.

The *Schistosoma japonicum*, Katsudura 1904, is found in the veins of the large intestine and also in the gastric, superior mesenteric, splenic, and cardiac veins. Both sexes are smaller than in the other two varieties, the male being 0.09 cm. in length, the female 1.2 cm., and the females are proportionately stouter than those of the other forms. The cuticle of the male is smooth, and the gynecophoric canal larger, and completely envelops the female. The ova are small, 75  $\mu$  by 45  $\mu$ , quite smooth, and have a rudimentary lateral knob, but no spine. The small cercariae enter the skin of man through hair-follicles and gland-ducts, and may give rise to some reaction on the surface in doing so. When the coupled worms have passed through the portal vein within the liver, they proceed thence to their ultimate destination in the intestinal venules. Here the ova are laid, and many are subsequently discharged into the intestinal canal through the mucous membrane, and may be discovered in the stools.

## SCIATICA

**Pathology.**—From an early stage the liver is enlarged, from a lymphocytic reaction followed by connective-tissue hyperplasia, owing to the irritation of innumerable ova carried to the organ from the portal vessels. Later an atrophic cirrhosis sets in, with thickening of the capsule and an irregularly nodular surface. The walls of the intestine are much thickened, and masses of eggs are seen therein; infiltration of mucous membranes followed by superficial erosions, or even deep ulcers, occurs, and sometimes a degree of sessile papillomaformation. The retroperitoneal and mesenteric glands are enlarged and firm. The spleen is greatly enlarged, both from portal stasis and from irritation of toxins, and shows a great increase of interstitial tissue. Ova may be deposited also in the brain and in other organs.

The **symptoms** of the earlier toxic effects are often very severe, and eosinophilia is pronounced. The established disease manifests itself in irregular attacks of chronic persistent dysentery, with fever, great enlargement of liver and spleen, and, later, general weakness and anaemia. This chain of symptoms may last from one to eight years, depending on the degree of infestation and reinfestation. In later stages all the evidences of severe portal obstruction declare themselves, and the patient dies from general exhaustion or intercurrent disease.

The **diagnosis** is fairly evident, and is confirmed by the discovery of ova in the faeces, though these often appear at irregular intervals. *Malaria* and other local diseases, such as *kala-azar* and *splenic anaemia*, must be excluded.

The **prognosis** is no better than in the other varieties, and **treatment** is essentially that of intestinal schistosomiasis: much is expected from the use of antimony intravenously on a large scale in the earlier stages.

FRANK COLE MADDEN.

### SCHÖNLEIN'S DISEASE (see PURPURA).

**SCIATICA.**—Sciatica, or sciatic neuritis, in the majority of instances is a perineuritis affecting the nerve in the neighbourhood of the sciatic notch. In some instances the nerve sheath and the nerve bundles may be damaged within the pelvis, and, again, the nerve may suffer from an inflammatory perineuritis below the level of the notch.

**Etiology.**—One of the commonest causes

of sciatica is rheumatism; the nerve sheath may become involved by a spreading inflammation of the fascial tissue from lumbago or from a lumbo-sacral fibrositis. Direct chill to the buttock may start this disease, and also injury by a fall or by prolonged lying in one position on the buttock. Toxæmias play a part in the causation of sciatica, both auto-intoxications as in gout and diabetes, and the toxins of such fevers as enteric, influenza, septicæmia, and many others. Disease of neighbouring joints, as chronic osteo-arthritis of the hip-joint, is a common cause of sciatica, the characteristic pain of which will then be superadded to the pain produced by the hip disease.

**Symptomatology.**—Usually the pain of sciatica spreads gradually from a lumbago down the back of the hip, thigh, and leg, to the front of the ankle, heel, and outside of the foot. This pain is generally increased by coughing and sneezing, and is often aggravated by standing, though in a small percentage of cases it is easier when the patient is moving about. The patient cannot lie on the affected side and usually keeps the limb slightly flexed at hip and knee. In some cases the pain is so acute that sleep is entirely prevented and doses of morphia alone give relief. Stretching the nerve by flexing the limb at the hip with the knee kept straight aggravates the pain considerably, and tenderness on pressure over the nerve at the notch or outside the tuber ischii is often met with. In a small proportion of cases, when the pain has persisted for several weeks or months, a deformity is produced by lateral flexion of the lumbar spine away from the painful limb, thus causing the hip of the affected side to be more prominent.

Examination of the reflexes shows no change in the knee-jerks, but the Achilles jerk on the affected side is frequently diminished or lost. The plantar reflex remains unaltered, though it may be diminished. The foot usually feels cold, and sweating is reduced. In the severer forms of sciatic neuritis, as when anæsthesia and motor weakness are present, vascular changes may be observed, such as swelling of the foot and ankle, and cyanosis with blotchy discoloration, increased by keeping the foot dependent.

**Diagnosis.**—Grosser lesions of the nerve, of the lumbo-sacral plexus, or of the spinal cord and spinal roots may simulate for a time the slighter form of neuritis known as sciatica, but the advent of motor paralysis, muscular wasting, and anæsthesia, or the appearance of a

## SCIATICA

proas abscess, or other disease of the vertebral column or of the spinal cord, will distinguish them from ordinary sciatica. In diabetic neuritis, and also in the neuritis due to direct exposure to cold, motor paralysis of the foot with muscular wasting and anæsthesia is not uncommon, though motor paralysis is extremely rare in the ordinary rheumatic type of sciatica.

**Treatment.**—In the acute stages, when there are great pain and tenderness, rest on an air-cushion or a water-bed is desirable. Immobilization by means of a long splint from axilla to foot is worse than useless, as change of position is necessary, patients being unable to remain constantly in one position. Various local treatments applied to the buttock and thigh may be of service, notably ionization, using a strong galvanic current. The electrodes should be large and pliable, and a thick layer of gamgee tissue or felt, thoroughly moistened, must be inserted between the metal electrodes and the skin. The negative electrode should be placed at the back of the buttock and should be moistened with 1-per-cent. solution of salicylate of soda. Lithium carbonate may be placed on the positive electrode. With large electrodes, measuring 6 in. by 8 in. or more, a current of 60 ma. may be used, and applied for twenty minutes daily. If the skin does not bear this daily treatment without the appearance of small acne spots, it is a good plan to give diathermy alternately with the galvanism. In some cases diathermy seems to do more good than the ionization; if so, it may be used daily instead.

Local radiant heat to the buttock and thigh sometimes gives relief, but only temporarily. Counter-irritation by blistering or by actual cautery along the back of the thigh over the sciatic nerve is at times of use. Massage or electric vibration should never be employed in the acute or subacute stage of sciatica, though at times both may be of use when chronic stiffness alone remains.

Forcible stretching of the nerve by flexing the hip with the knee straight—a treatment much in vogue with some Swedes—is scientifically wrong and usually aggravates the pain. Worse still is forcible stretching of the nerve by a surgeon after cutting down and exposing the nerve in the buttock. The adhesions in chronic sciatica between the nerve bundles and the sheath require to be burst asunder laterally and not by longitudinal traction. This, effected by scarifying the nerve, splitting the nerve bundles longitudinally and scarifying

## SCLERA, AFFECTIONS OF

them, sometimes cures obstinate chronic cases in which numbness and anæsthesia of the foot and loss of the Achilles jerk indicate a chronic damage of the sciatic nerve. Before this drastic procedure is attempted it will be well to try saline injections into the nerve, 60 to 80 c.c. of the solution being injected within the sheath of the nerve: when properly done this is sometimes brilliantly successful; it is a procedure, however, which requires some technical skill and practice. A simpler injection treatment is that of air, a cushion of air being injected in the neighbourhood of the nerve, not into it; the injection may be repeated three or four times. Hypodermics of morphia or heroin are sometimes necessary daily, or even oftener, to procure rest and sleep, but particular care should be taken to avoid the induction of a drug habit in these cases. As soon as the pain shows any signs of abating, and the patient is able to get about again without much suffering, aspirin, phenacetin, or pyramidon should replace the morphia. A course of baths at Droitwich or some other spa is a useful means of completing the cure.

WILFRED HARRIS.

**SCLERA, AFFECTIONS OF.** The diseases here considered are Episcleritis, Scleritis, Tumours, and Staphyloma.

**Episcleritis.**—A subacute inflammation involving the subconjunctival tissues and the superficial layers of the sclera.

**Etiology.**—The disease attacks young adults, especially those with a "rheumatic" tendency. The removal of carious teeth or septic tonsils, the treatment of pyorrhœa, nasal sinusitis, constipation, salpingitis, etc., may banish the symptoms and so establish the etiology.

**Symptomatology.**—Only part of the anterior portion of the sclera is affected, forming a vascular patch with its centre some distance from the corneal margin. This area is congested, with a purplish rather than a red tint, since the dilated vessels are well beneath the surface (this is a point of distinction in the diagnosis from a localized conjunctivitis). Episcleritis is attended with little pain or general disturbance, but there is a certain amount of aching in the eye, and a disability from reading for any length of time. There is practically no discharge from the conjunctiva. The inflammation is often transient and recurring, the attacks lasting from a few days to some weeks. In other cases it is more persistent, and there may form at the centre of the patch

## SCLERA, AFFECTIONS OF

a small *scleral nodule*; this is at first red and later yellow in colour, coincident with the formation of a small bead of pus at the centre. Such a nodule is adherent to the scleral tissue and may have formed round a minute foreign body.

**Treatment.**—Locally, little can be done beyond relieving the congestion by the application of hot bathings and fomentations, irrigation of the conjunctiva by boric-acid lotion, and some gentle massage with boric or weak yellow oxide of mercury ointment. If a scleral nodule forms, it may be incised with the point of a sharp knife. Mixtures containing salicylate of soda are of value.

**Scleritis** is nearly always associated with iritis, cyclitis, or keratitis. It is a chronic persistent inflammation in which the whole of the ring of sclera around the cornea is involved; this zone shows a dull purplish colour, with some large dilated vessels beneath and in the conjunctiva. Signs of iritis and cyclitis in the form of posterior synechiæ, corneal deposits (*keratitis punctata*), and vitreous opacities will be found on examination, and poor vision will be complained of. Interstitial keratitis, when severe, often causes scleritis in its later stages, but there is also *sclerosing keratitis*, in which, following a marginal ulcer, the cornea becomes converted into tissue resembling sclera. A certain number of cases of scleritis are of tuberculous or syphilitic origin, but the cause is often difficult to determine.

**Treatment.**—The local measures will in the main be similar to those used in episcleritis, but the associated intra-ocular disease will also require treatment, e.g. atropine for irido-cyclitis. Blisters applied to the temple often give temporary relief.

**Tumours** sometimes appear on the outside of the sclerotic, in connexion with perforating growths from the interior of the globe, e.g. sarcoma of the ciliary body, glioma of the retina. Occasionally a gumma or tuberculous mass from the ciliary body may ulcerate through.

**Staphyloma** of the ciliary region is a stretching of the sclera external to the limbus and iris zone. It is usually the result of increased intra-ocular tension (glaucoma) of long duration. After excision of a blind eyeball a staphyloma in the equatorial region is not uncommonly found. Clinically, a ciliary staphyloma appears as a localized bulging of the sclera; it is of a slate colour, owing to the

## SCLERODERMIA

visibility of the underlying pigment through the thinned tunic. Treatment is not indicated, unless the eye is painful, when it may be excised.

In high myopia the posterior half of the sclera is stretched, and this may form a *true posterior staphyloma*, visible with the ophthalmoscope as a dark shadow on the fundus, to the nasal side of the optic disc.

F. A. JULER.

**SCLEREMA NEONATORUM.**—This name should be restricted to a condition in which, within a few days of birth, a waxy, rigid change occurs in the integument. The infant so affected has a subnormal temperature, its functions are interfered with, and, as a rule, death rapidly supervenes. (Edema neonatorum (*see* EDEMA) is sometimes mistaken for sclerema, and, like the latter, is usually soon fatal; but here a condition of solid oedema of the skin and subcutaneous tissues occurs.

H. MACCORMAC.

**SCLERITIS** (*see* SCLERA, AFFECTIONS OF).

**SCLERODACTYLIA** (*see* SCLERODERMIA).

**SCLERODERMIA.**—A condition of hardness or rigidity of the skin, caused by an overgrowth of fibrous tissue. It is met with in two different forms—(1) generalized sclerodermia, (2) localized sclerodermia, or *morphea*.

**Etiology.**—Nothing is known of the cause. Sclerodermia is chiefly a disease of young adult life, and is about three times as common in women as in men; this is particularly true of the localized form. It has been attributed to a tropho-neurosis, to alterations in the secretions of the endocrine glands, especially the thyroid, to an endarteritis, and to a primary hyperplasia of the collagen fibres of the skin, but no definite evidence is forthcoming to clear up the question.

**Pathology.**—The chief histological changes noted are an overgrowth of the collagen bundles in the corium and subcutaneous tissue, with replacement of fat by fibrous tissue in the latter; and an exudate of cells around the vessels of the corium, together with some endothelial proliferation. The epithelium is usually unaffected, but is sometimes flattened by pressure of the hypertrophied corium, and in some cases shows an excess of pigment cells, which are also present in the corium.

**Symptomatology.** (1) **Generalized sclerodermia.**—The symptoms may appear rapidly or slowly. In both cases a disturbance of the

## SCLERODERMIA

health, such as fever, pains in the joints, neuralgia, or itching may precede or accompany the attack, but this is not by any means the rule. Stiffness of the parts involved is often the first symptom, and may spread rapidly or slowly, until it produces fixity of the joints, followed by progressive wasting of the muscles; breathing may become difficult owing to fixation of the skin of the chest; and the taking of solid food may be prevented by the rigid condition of the cheeks and mouth. The skin appears swollen and glossy, and is very hard; the deeper structures are fixed and the furrows of the skin disappear. The colour is not appreciably changed, though it may be paler and more waxy than normal. The lesions begin symmetrically, and generally in the upper part of the body. In some cases the mucous membrane may be involved. In one form, *sclerodactylia*, the affection attacks the fingers and hands. The skin is drawn tight over the fingers of both hands, fixation of the joints occurs, and later, atrophy sets in so that the fingers become pointed and the skin appears to be drawn tight down on to the bones.

(2) **Localized scleroderma (morphœa).**—Patches of scleroderma occur which may vary from the size of a pea to large patches involving almost all the back or front of the trunk. The patch may be pinkish in colour and raised from the surface, with smooth polished surface and rigid feel: or it may be of old-ivory colour, level with or slightly depressed below the surface of the skin, very rigid and fixed, and often surrounded by a lilac-coloured border; or, again, it may be dead-white, slightly depressed, with more or less irregular edge and of normal consistence. This last type may sometimes show much scaling. Patches may occur in any part of the body, the legs and trunk being most frequently affected. The patches may vary considerably in number; usually they are single or few, but sometimes they are very numerous. One type, in which a large number of small patches occurs, is a form of so-called "white-spot disease." Another type is the "band" form, in which long strip-like lesions are present.

**Diagnosis.**—The generalized type is characteristic, but must be distinguished from *sclerema neonatorum*, which is a condition of solidity of the subcutaneous fat and occurs in the first few weeks of life, while scleroderma does not occur till later.

The white morphœa patches can be distin-

## SCOTOMA

guished from *leucoderma* by the absence in the latter of any other than pigmentary change in the skin and by the presence of a hyperpigmented zone around.

**Prognosis.**—The more severe forms often end fatally, but the milder generalized cases and the localized variety sometimes clear up spontaneously. Often, however, they last for many years.

**Treatment.**—In the generalized cases the use of warm clothing to prevent excessive loss of heat, and massage, are indicated, while cod-liver oil given internally is the most useful remedy. In all types thyroid extract has been used largely, but is of doubtful value. Thio-sinamin injected locally is sometimes of value in morphœa. Treatment, however, is usually unsatisfactory.

A. M. H. GRAY.

**SCLEROSIS, CEREBRAL** (*see CEREBRAL SCLEROSIS*).

**SCLEROSIS, DISSEMINATED** (*see DISSEMINATED SCLEROSIS*).

**SCLEROTIC, AFFECTIONS OF** (*see SCLERA, AFFECTIONS OF*).

**SCLEROTIC, EXAMINATION OF** (*see EYE, EXAMINATION OF*).

**SCOLIOSIS** (*see SPINAL CURVATURE*).

**SCOPOLAMINE POISONING** (*see POISONS AND POISONING*).

**SCORBUTUS** (*see SCURVY*).

**SCOTOMA.**—This term signifies the loss or sensible diminution of vision in any part of the visual field, but is most commonly applied to loss in the central portion of the field and to defects separated from the periphery by areas of retained vision; we speak, for instance, of a central scotoma, or a ring scotoma, or an island scotoma. A scotoma may be absolute or relative; it may be absolute for colours and relative for white, or absolute for some colours and relative for others, or there may be no scotoma discoverable for white though a relative scotoma for colours exists. In certain retinal and choroido-retinal diseases, positive scotomata occur owing to retinal irritation producing sensations of colours, or flashes referred to the blind or affected area. The central scotoma of acute retrobulbar neuritis is one of the most characteristic forms met with. It may be either small or extensive; in very acute cases it may reach to the

## SCURVY

periphery, so that complete blindness is produced. Central scotomata relative for white and absolute for colours, more rarely absolute for white also, form the most characteristic symptom of the toxic amblyopias. Retinitis and choroido-retinitis produce scotomata corresponding to the areas affected; their occurrence is the best guide to the investigation of the fundus in the early and more obscure stages of these diseases. In primary atrophy of the retina (retinitis pigmentosa) a ring scotoma, partial or complete, is common, and a similar but more irregularly shaped ring made up by the fusion of island scotomata accompanies the secondary atrophy of the retina occurring in disseminated choroiditis. A similar ring scotoma is frequently present in Leber's familial atrophy. The careful investigation of scotomata in the visual fields due to lesions of the optic nerves, chiasma, tracts, optic radiations, and occipital lobes is of the greatest importance as an aid to the localization of intracranial lesions. Reference to the writings of Cushing on pituitary disease and of Holmes on the cerebral localization of vision will enable readers to appreciate the value of the results which have been achieved by careful work in mapping out scotomatous areas.

LESLIE PATON.

**SORIVENER'S PALSY** (*see* NEUROSES, OCCUPATION).

**SCROFULODERMIA** (*see* SKIN, TUBERCULOSIS OF).

**SCURVY.**—"The scurvy had begun to show itself on board. . . . His legs swelled and pained him so that he could not walk; his flesh lost its elasticity, so that if it was pressed in it would not return to its shape; and his gums swelled till he could not open his mouth. His breath, too, became very offensive; he lost all strength and spirit, could eat nothing, grew worse every day; and in fact, unless something was done for him, would be a dead man in a week, at the rate at which he was sinking." This description of the effect of scurvy upon a young and previously healthy seaman was written by a man who had no special knowledge of medicine, and gives in short compass a picture of the disease not easily bettered. The sole addition which need be made is to mention the tendency to subcutaneous hæmorrhages. We may add a further word from Dana's volume. "The medicines were all, or nearly all, gone; and if we had had

a chest full they would have been of no use; for nothing but fresh provisions has any effect upon the scurvy." Onions and potatoes were secured from a passing brig, and ten days afterwards, so rapid was the patient's recovery that "from lying helpless, and almost hopeless, in his berth, he was at the mast-head furling a royal."

**Etiology.**—It is therefore the want of certain elements contained in fresh food, especially vegetable food, that produces these remarkable phenomena. Hence, in modern days, when long voyages without a call at port are exceptional, and when the means of avoiding the disease are better known, scurvy in the adult is a rare disease. It is seen now and again in seamen who have served in a sailing ship detained beyond expectation by winds and storms; it is one of the dangers which beset the Arctic and Antarctic explorer; it appears now and then in lumber and mining camps; it devastated the bands of Indian coolies who built the Uganda railway; and it occasionally attacks persons who from poverty or from dyspepsia have condemned themselves to starvation diet badly selected.

In civilized communities it is now most often seen in young infants, and presents a peculiar and easily recognized picture, though one the significance of which, as is clear from the variety of names given to it, long eluded medical science. Acute hæmorrhagic periostitis, acute rickets, and hæmorrhagic rickets were the names given before the work of Cheadle and Barlow revealed the true meaning of the symptoms and caused it to be generally regarded as "infantile scurvy." The designation "scurvy rickets," which is often employed, is misleading and should be abandoned; for though rickets is often present, it is not the cause of the chief phenomena.

There have been many hypotheses as to the nature of scurvy. Some have regarded it as an infection, but the evidence is entirely unconvincing; almost the only feature which resembles those of an infective disease is the selective character of the attacks. Of a crew of considerable numbers, living on the same food and in the same conditions, but two or three will suffer from the disease. The idea of a specific infective agent can be dismissed. A second hypothesis, that it is due to the presence of toxins in the foods consumed, much of which is tinned or otherwise preserved, has more to recommend it; but the extraordinary effect of any fresh vegetable

## SCURVY

food, or fruit or vegetable juice, upon the symptoms points very strongly to the truth of a third hypothesis, that the disease is due to the absence of certain elements, necessary to healthy life, from the food consumed. Recent knowledge acquired of the importance of vitamins confirms this view, and it is now established that the cause of scurvy is the absence from the food of an accessory food factor. The addition to the scurvy-producing diet of small quantities of foods known to contain this antiscorbutic factor protects completely from the disease.

**Morbid anatomy.**—In the bodies of those dead of the disease there are generally no marked peculiarities other than those associated with severe grades of anæmia—for example, petechiæ in the serous membranes and in the muscles, and a universal œdema of the tissues. In scurvy of the infant, however, the symptom which attracts the chief attention in life is due to the phenomenon which attracts instant attention after death; that is, the extravasation of blood beneath the periosteum of the large bones, or ribs, or occasionally of the skull and face. In a typical instance the periosteum is stripped completely from the shaft of the femur and the intervening space is filled with extravasated blood, which in the cases of longest standing has clotted and begun to be replaced by new tissue, including young periosteal bone.

**Symptomatology.**—There is little to add to Dana's striking description. The earliest symptoms are increasing languor and pallor, and marked anorexia; the gums swell early, and soon become "spongy," and bleed easily, while the teeth show a tendency to be loosened and fall out. The skin is rough, the extremities are œdematous, with petechiæ beneath the skin; very occasionally bruises may ulcerate. A slight degree of hæmaturia is not uncommon; other hæmorrhage is rare. Remarkable ocular phenomena sometimes occur; one man may lose his vision during the day, another be quite unable to see things after dusk (hemeralopia, nyctalopia).

**Symptoms of infantile scurvy.**—These are in some respects, though not in essentials, so different from those seen in the adult that they must be described separately. In the first place it must be recognized that the victims of infantile scurvy are in the vast majority of cases those who have been fed on patent and proprietary foods. Scurvy occurring in the breast-fed, or in infants fed on cow's milk

even when boiled, is a rare event, and, unless very well authenticated, such cases should be regarded with suspicion. I have seen but one case in an infant fed on boiled cow's milk, and none in breast-fed children; of the many others, the great majority were in infants fed on proprietary infant foods, unguarded by the administration of fresh fruit or vegetable juice. The child becomes pale, irritable, loses weight, and refuses food. Presently he refuses to move his limbs, and if these are touched, screams with pain; after a day or two he is apt to scream even at the approach of the doctor who will handle his limbs. At this time, if he has any teeth erupted the gums about them will show a purplish tint and will bleed readily. The motionless limb on being handled is found to have a swelling, usually at the diaphyseal end of the bone; the least pressure on this swelling is painful, and motion of the neighbouring joint cannot be tolerated. As a rule, there is no effusion into the joints; but there is often an œdematous condition of the skin and subcutaneous tissues over the periosteal swelling. These periosteal swellings are most easily palpable at the lower end of the femora, at the ankles, and in the shafts of the humeri, but occur in all the long bones, and often also in the ribs. Another curious, but infrequent, symptom is protrusion of the eyeball on account of orbital hæmorrhage. There are also occasionally flat extravasations beneath the pericranium covering the bones of the cranial vault. Petechiæ and bruises are common, as in the adult; hæmaturia in slight degree is also usual, but may easily be missed unless special inquiry is made. Fever is very slight or absent, save in the cases complicated by severe infective diarrhœa or broncho-pneumonia.

A curious symptom described by Barlow in his original papers on the disease is the flattening of the thorax; the long ribs appear to be straightened out, and to form an angle at their juncture with the cartilages, which approaches in severe cases to a right angle, so that the sternum and cartilages present a flat surface marked at its outer margins by the prominent ends of the bony ribs.

**Course.**—In either adults or infants the course of the disease depends upon the detection of the cause of the symptoms, and the early application of the known means of remedy. If either the disease be not recognized or no efficient remedy can be obtained, the patient will die from gradually increasing weakness and anæmia, complicated often with diarrhœa



## SCURVY

or pneumonia. When once put under the requisite conditions of diet he makes a recovery of remarkable rapidity. A proper revision of the diet is usually followed by a remission of all the urgent symptoms within forty-eight hours, and a rapid return to a healthy condition of the tissues and blood.

**Diagnosis.**—In **adults** there is, as a rule, but little difficulty in recognizing the disease, provided the possibility of its occurrence remains in the mind of the physician; error arises chiefly from the failure to recognize that increasing weakness, anæmia, and œdema of the lower extremities may be due solely to deficiencies in the diet. In some instances the hæmorrhagic tendency of the complaint is sufficient to suggest the diagnosis of *purpura hæmorrhagica*; but this disease is, as a rule, of sudden, even of abrupt onset, and seldom presents the combination of debility, œdema, and pallor at the beginning of the illness. Moreover, in cases of scurvy, hæmorrhages from the mucous surfaces other than the gums are of rare occurrence, though common in *purpura*.

In the **infant** the disease is much more easily overlooked. If there are no teeth the gums often remain healthy, and the symptoms are limited to fretfulness, tenderness of the limbs, and pseudo-paralysis or absence of voluntary movements on ordinary stimulation. In such cases the swelling around the joints may be mistaken for that of *epiphysitis*, either acute or syphilitic. Acute epiphysitis is limited to one joint, whereas the effusions in scurvy are nearly always multiple; scurvy is usually afebrile, or at most shows a moderate fever, while in acute epiphysitis the fever ranges high; in scurvy there is little or no leucocytosis, in acute epiphysitis the leucocytes are considerably increased in number. Syphilitic epiphysitis occurs almost invariably before the age of 4 months, usually at from 6 to 12 weeks of age; scurvy is rare before 6 months of age. In this difficulty an X-ray picture of the swelling will show, on the one hand, the characteristic features of syphilitic periostitis; while the scurvy picture shows the subperiosteal effusion. In some instances the question arises whether the swelling is due to a *sarcoma of the bone*; it can only be answered by careful inquiry as to the history of the swelling, and occasionally even only by the result of dieting. Operative exploration for diagnosis is unjustifiable.

There are cases which, whilst they afford

no definite evidence of scurvy, exhibit the tenderness to the handling of the limbs, combined with anorexia and increasing pallor. Such infants must be regarded as scurvy patients in the early stages; and appropriate treatment will, in the majority of cases, amply confirm the diagnosis.

In conclusion, the physician must always be quick to suspect the existence of this disease in every infant who, being between the ages of 6 and 15 months, and fed on an artificial diet, develops an extreme dislike of movement, whether the classical signs of the disease are present or not.

**Treatment and prognosis.**—The results of treatment are astonishing. The addition to the diet of fresh fruit, or vegetable juices, will alter completely the appearance, the feelings, and the outlook of the patient in the course of forty-eight hours. All who have seen scurvy in the adult speak of the extreme depression which characterizes the illness; and in the infant the fretfulness and cries of pain are equally impressive. These symptoms disappear within forty-eight hours; the adult becomes cheerful, the infant calm and restful. The swelling and œdema of course remain longer, but even their disappearance is extraordinarily rapid. To attain these results it is only necessary to add to the diet the juice of fresh fruits, or that of raw vegetables, potatoes or onions; from some experiences it would appear that fresh meat partially cooked or raw meat juice has a similar though less prompt effect. In the infant it will suffice to add a little steamed potato to the milk in the bottle, and 3 or 4 teaspoonfuls of lemon or orange juice each day. Similar measures should be adopted for prophylaxis. Lemon juice was long employed in the navy and mercantile service on all long voyages, and its use practically abolished the disease from these services. The complications to be treated are few; in the adult attention must be paid to the mouth to avoid the risk of septic complications; and in the infant the affected limbs should be carefully splinted or bandaged to avoid any risk of fracture of the softened bones. In the worst and most neglected cases diarrhœa may set in with the addition of the fresh food, and may prove fatal; but, considering the gravity of the condition in severe examples and the extraordinary quickness of recovery, it may certainly be said that in no other disease can the patient approach so near the gates of death and yet be rescued.

HUGH THURSFIELD.

## SEBACEOUS CYSTS

**SEA-SICKNESS** (*see* Reflex Vomiting, under STOMACH, FUNCTIONAL DISORDERS OF).

**SEBACEOUS CYSTS.**—Retention cysts of the sebaceous glands, due to dilatation either of the duct or of the gland itself. They do not occur before the adolescent age, and while they may develop wherever sebaceous glands are present, they are found most frequently on the scalp, face, neck, shoulders, back, or scrotum. They have a fibrous wall lined by stratified squamous epithelium, and the contents are composed of the retained sebaceous material.

Sebaceous cysts are of very slow growth. The smaller ones form hard, white, beadlike nodules projecting from the surface; the large ones vary much in size, up to that of a walnut or even larger. Sebaceous cysts are rounded, well defined, and have smooth surfaces. They are attached to the overlying skin, but are freely movable upon the deeper structures. There is no pain unless suppuration has occurred. The orifice of the gland-duct, blocked by a comedo, can often be recognized, and the contents may sometimes be expressed from it. The consistency varies; the cysts may have a pulsatious feel or may distinctly fluctuate. They are apt to be multiple.

Inflammation and suppuration are common. On the scalp, rupture of the abscess may be followed by prolific growth of granulations, so that malignant disease is simulated. Malignant transformation does occur, but very rarely. Calcification of the cyst-wall and its contents may convert the whole tumour into a calcareous mass.

**Treatment.**—This consists in removal, which must be complete, or recurrence will take place. It is better not to attempt to remove the cyst intact. An incision should be made into it, freely opening the cavity, and the contents carefully removed. The wall of the cyst can then be separated on each side from the margin of the incision, and traction on it will usually drag it away entire. The wound is sutured without drainage.

When suppuration occurs it is best to incise the cavity and to postpone excision of the cyst-wall until the inflammation has subsided and the wound is healed.

Sometimes, owing to previous attacks of inflammation, the wall of a sebaceous cyst will not peel out from its bed. In these circumstances as much as possible of the con-

## SEBORRHOEA

tained wall is dissected out and the remainder curetted away. The cavity is painted with pure phenol. A drainage-tube must be left in for forty-eight hours.

C. A. PANNETT.

**SEBORRHOEA.**—There is some difference of opinion as to the exact application of this term, but here it is used to denote a condition in which there is an excessive sebaceous secretion; in fact, it is used in the sense in which the older term "*seborrhœa oleosa*" was used.

**Etiology.** The condition is most common in adolescence. It begins with the increased development of the glandular structures in the body which occurs at puberty, and is most active at that period, gradually diminishing in the healthy subject after the age of 30 or so. It appears to favour certain families and races, being especially common in some of the dark-skinned races. Some persons suffer throughout life, but they are usually those whose health and habits are not quite normal. In those who suffer from gastric disturbances, constipation, anæmia, uterine troubles and the like, or who habitually over-eat, consume too much alcohol, smoke excessively, and possibly in those who indulge in sexual excesses, the condition tends to persist.

**Pathology.**—Seborrhœa appears to be a functional disturbance of the sebaceous secretion which is usually, if not always, accompanied by a similar increase in sweat secretion. Some have considered that micro-organisms play a part in its production, but the general opinion is against this view.

**Symptomatology.** The excessive secretion is chiefly noticed on the face, where the sebaceous glands are highly developed, particularly on the nose and in the region of the nasolabial furrows; it is also present, however, on the scalp, in the concha of the ear, on the sternal region of the chest, and on the back. The skin in these regions is shiny and covered with a thin layer of oil. The skin itself appears thick and of a muddy-white colour, and the mouths of the sebaceous follicles are patulous and very prominent. Acne vulgaris, rosacea, and seborrhœic dermatitis are frequent complications. The hair of the scalp is limp and greasy.

**Treatment.**—The general health must be attended to. Constipation, dyspepsia, and anæmia especially require correction. The food should be regulated, excess of starches and sugars being avoided; alcohol and smoking

## SEBORRHOIC DERMATITIS

should be kept within moderate limits, and the patient should be encouraged to take plenty of out-of-door exercise. Locally, washing with soap and water at frequent intervals, followed by the application of a sulphur (5 per cent.) and talc powder, is sufficient for the milder cases. In the more severe cases in which the follicles are plugged with sebum the more drastic remedies indicated for acne vulgaris (q.v.) are recommended. A. M. H. GRAY.

**SEBORRHOIC DERMATITIS.**—Under this heading are included an inflammatory condition of the scalp which shows itself by the formation of "scurf" or "dandruff," and was formerly known as "seborrhœa sicca"; and certain inflammatory dermatoses which occur on other parts of the body, chiefly on the face, chest, and back, and are characterized by the presence of more or less circumscribed reddish patches covered with greasy scales.

**Etiology and pathology.**—The vagueness of the above definition is due to uncertainty as to the etiology of the conditions. The endeavour is to limit the term to certain eczematoid eruptions of bacterial origin, but although clinically there is every reason to suppose that the conditions indicated have this origin, yet the evidence as to the particular organism concerned is not conclusive. Three organisms are constantly found in the lesions of seborrhœic dermatitis of adults, the acne bacillus, the bottle bacillus (or spore of *Malassez*) and a white staphylococcus; the first is said to be generally absent in cases occurring before puberty. The lesions of seborrhœic dermatitis have some resemblance to those produced by certain ringworm fungi, and it is therefore not a far-fetched assumption that the bottle bacillus, which is a yeast-like organism found universally in such lesions, may be the producing agent. This view is supported by the work of Sabouraud and others, but is not by any means universally accepted.

Not only do the specific lesions of seborrhœic dermatitis occur in seborrhœic subjects, but such persons are prone to develop ordinary eczema (that is, a non-bacterial dermatitis), and are very liable also to impetigo contagiosa. In both of these affections the lesions may be distributed in the same manner as in genuine seborrhœic dermatitis, and in both also there may be greasy scales and crusts, owing to the fact that the skin is abnormally greasy. It is owing largely to this circumstance that so

much confusion as to what should be termed seborrhœic dermatitis has arisen.

The morbid anatomy is characterized by a cellular infiltration in the superficial layers of the dermis, proliferation of the mucous layer and an irregular formation of the horny layer, between the cells of which may be found collections of leucocytes. The pronounced œdema of the epidermis with vesicle-formation found in eczema is absent. It must be noted, however, that the changes indicate a low grade of inflammation similar in nature to that found in eczema.

**Symptomatology.**—Seborrhœic dermatitis of the scalp is the commonest form. It may be said to be almost universal in this country, though it varies much in degree. It is characterized by the presence of fine branny scales situated on the scalp and around individual hairs. As a rule, no redness of the scalp is present, nor indeed anything to suggest an inflammatory process. In many cases the process is so slight as to cause no inconvenience, but often the scales are larger, more frequently renewed and more greasy, and the patient is incommoded by these scales falling on his clothes. Much more rarely they form thick greasy masses overlying the whole scalp.

The persistence of this condition, even in its mildest form, is thought to be responsible for loss of hair, and especially for the so-called *alopecia præmatura*, which is characterized by the recession of the hair from the forehead and thinning and eventual baldness spreading from the crown of the head. It is probable, however, that other factors also play a part in this condition.

The earliest stages of this scurfy condition are seen in young infants, who develop reddish scaly rings on the scalp in which the bottle bacillus can be found; these spread, and the redness disappears, but the infection, which was presumably caught from the parents, does not die out but persists in the form already mentioned.

Occasionally the chronic scaly condition becomes acute, the scalp becomes irritable, red, and tender, and thick crusts may form. The redness and crusting may spread beyond the margin of the hairy scalp.

The face may also be affected, especially the eyebrows, forehead, naso-labial folds, beard and mastoid regions. The lesions here are dry, reddish, or pale patches surmounted by greasy scales or crusts. On close examination it can usually be seen that the lesions are follicular in

## SEBORRHŒIC DERMATITIS

origin and that the patches are formed by an aggregation of these follicular lesions.

The ears also may be affected, especially the concha, and some forms of blepharitis appear to be of seborrhœic origin. An intractable scaly inflammation of the lips, *cheilitis exfoliativa*, is also said to be due to the same cause.

On the chest and back, ring or figurate lesions are most common. On the chest the lesions are generally localized to the skin over the sternum, and they may take the form either of superficial follicular papules or of circinate and figurate patches. On the back the lesions begin in the interscapular region and may spread over the whole back.

Occasionally similar lesions are found among the pubic hairs.

Of certain other types of dermatitis that have been included under this title, the claim to be true examples of seborrhœic dermatitis is not by any means secure. Thus circumscribed, pinkish or red, circular or oval patches, covered with very fine dry branny scales, are met with on various parts of the trunk and limbs. They are usually single or few in number, but occasionally numerous. The scales have no characteristic flora. If very resistant to treatment, they are classed with the parapsoriasis, but if they respond to treatment, and especially to sulphur, the tendency is to call them seborrhœic. To this type belong the chronic scaly patches, often becoming moist because of their situation, which are found, in many cases symmetrically placed, in the flexures of the body, axillæ, elbows, groins, and knees.

There is also a group of cases closely resembling psoriasis at first sight, but in which the scales are not dry as in psoriasis but greasy and coarser, and when removed leave a moist surface. These may occur in isolated patches, or may be very numerous and extensive. Their etiological position is not by any means clear.

**Diagnosis.**—Seborrhœic dermatitis of the scalp in children must be differentiated from ringworm. The scaly rings seen in infants in the former affection show the bottle bacillus but no mycelium. In older children a scurfy scalp requires very careful investigation to determine the presence or absence of ringworm stumps. In certain cases, especially of the endothrix variety of ringworm, the typical ringworm patches are not present, but only here and there are stumps found, and these cases often show an abundance of scurf. With perseverance, however, stumps can be found which show the offending fungus.

In acute cases the diagnosis from *impetigo contagiosa* has to be made. The presence of scattered impetigo vesicles or crusted lesions, or of that forerunner of impetigo, pediculosis capitis, may help to settle the diagnosis; but as simple scratching may determine an attack of impetigo on a seborrhœic scalp, the diagnosis between a simple impetigo and an impetiginized seborrhœa is not always easy.

On the face the same difficulty arises: a simple impetigo is very apt to distribute itself like a seborrhœic dermatitis, or the latter may develop a secondary infection. The history, the presence of impetigo lesions on other parts of the body, and the response to treatment, will give help in arriving at a diagnosis.

From eczema the diagnosis is generally made by its more extensive distribution, and by the involvement of the cheeks and eyelids; but the two conditions may coexist. Greasiness of the scales is not always a diagnostic feature, as eczematous scales in a seborrhœic patient will of course be greasy.

The figurate lesions on the trunk may be differentiated from ringworm and *pitryiasis versicolor* by the presence of mycelium in the scales of these two conditions. The circumscribed cases with branny scales have to be separated from *pitryiasis rosea*. In the latter affection there is a more or less acute onset, and the lesions present characteristic features—a smooth raised pink periphery, with a collarette of fine scales inside, and within this a pale fawn-coloured centre. From parapsoriasis the diagnosis is made by the extreme chronicity of this condition and its failure to respond to treatment. The differentiation of the psoriasiform type from psoriasis has already been touched upon.

**Prognosis.**—Seborrhœic dermatitis tends to respond well to treatment, but lesions are very apt to recur, and it appears to be impossible, in the existing state of knowledge, to stamp out the infection. This especially applies to the scalp. Treatment, therefore, has generally to be continued for long periods. Some acute cases, especially in children, are very resistant to remedial measures.

**Treatment.**—Sulphur appears to be almost a specific for this condition. The figurate forms on the body are rapidly removed by an ointment consisting of 3 per cent. each of sulphur and salicylic acid in a soft paraffin base. So definite is its action that it is often used as a means of diagnosis.

## SEBORRHŒIC DERMATITIS

This treatment is also of equal value in the less acute cases where the *face* is involved, but it is not so satisfactory in the acute cases, nor when secondary infection has occurred. In the impetiginized seborrhœa of the face, removal of the crusts with warm water or oil is the first procedure, to be followed for a day or two by the application of a 1-in-1,000 acriflavine lotion. When the oozing has ceased, calamine liniment (B.P.C.) with 5-10 per cent. ichthyol should be applied, and later, if necessary, sulphur and salicylic acid, 3 per cent. of each, in a cold cream may be used.

The treatment of seborrhœic dermatitis of the *scalp* is a more troublesome procedure and requires to be carried on for some time. The scalp should be frequently washed either with spirit soap (spirit. sapon. kalin. (B.P.C.) being one of the best) or with extractum quillaæ liq. (B.P.C.). Medicated soaps, such as sulphur or tar, are preferred by some. Lysoform, a liquid formalin soap, is also very useful, but should not be used too frequently owing to its irritating effect. The frequency of washing will depend upon the degree of scurfiness; in men it is often advisable to recommend it daily, but in women once a week is about the maximum that can be expected.

In milder cases lotions are usually sufficient, and are pleasant for the patient to use. Resorcin is one of the most useful drugs for this purpose, but owing to its tendency to stain white or blonde hair, it should only be used by dark-haired patients. A useful formula is resorcin 1 2 dr., sp. coloniënsis 1 oz., water to 8 oz. If the hair is very dry, ol. ricini 1-2 dr. may be added. Acetic acid  $\frac{1}{2}$  oz. is another useful adjunct. As a substitute for resorcin, chloral hydrate 1 or 2 dr., or perchloride of mercury 2-4 gr., may be named.

In all cases where there is much scurfiness, it is well to begin treatment with an oil or an ointment. For ointment the formula recommended by Whitfield, of paraffin molle 2 dr., ol. coco. nucif. to 1 oz., forms an excellent basis for the application of medicaments to the scalp. Of these the most useful are salicylic acid and precipitated sulphur, 15 gr. of each. Resorcin 15 gr., thymol 15 gr., ammoniated mercury 15 25 gr., or oil of cade  $\frac{1}{2}$  oz., may also be used.

If the scalp is inflamed the washing with soap should be dispensed with and the scales and crusts removed with warm water or olive oil. If there is much crusting, starch poultices may be required. Mild antiseptics, such

## SEMILUNAR CARTILAGES

as 1-in-4,000 perchloride of mercury, or 2.5 per cent. ammoniated mercury in linimentum calcei, should be used in the first instance, followed by the lotions or ointments mentioned above, if required.

The dry branny patches on the trunk and limbs and the psoriasiform patches often require stronger remedies, such as tar, pyrogalllic acid, chrysarobin. The flexural cases are very resistant to treatment, and the lesions should be tied up to prevent scratching. A zinc paste containing 5-10 per cent. of tar suits best; considerable relief may be given by X-rays or phototherapy. A. M. H. GRAY.

**SEMILUNAR CARTILAGES, DISLOCATION OF.**—Displacement of one or both of the semilunar cartilages of the knee-joint, as a result of injury.

**Etiology.**—The condition usually affects young men, and is due to an injury with the joint in a position of semiflexion, most commonly sustained on the football field or in the gymnasium; pre-existent laxity of ligaments is a predisposing cause.

**Pathology.**—The *internal* is very much more commonly injured and dislocated than the external semilunar cartilage; the former is firmly attached at its convex margin to the deep part of the internal lateral ligament and capsule of the knee. As a result either of direct violence applied to the outer side of the joint, or of a twist, the knee being in semiflexion, part of the fibres of the internal lateral ligament are detached from the tibia, and the internal semilunar cartilage is displaced inwards, the attachment of its anterior horn being ruptured or stretched, or the cartilage itself being fractured. At the time of the injury the internal condyle of the femur is separated from the internal tuberosity of the tibia so that the inner part of the joint is "open"; when the violence has spent itself the inner part of the joint closes again, and the cartilage either slips back into its normal position, or more commonly is caught between the two bones, causing "locking" of the joint, which cannot be fully extended. The lesion of the cartilage may take a variety of forms: it may be loosened or detached anteriorly, ruptured transversely or obliquely, split longitudinally, rolled up, or even completely detached. The injury is followed by synovial effusion, which under treatment clears up in about a fortnight. Recurrent attacks of dislocation of the cartilage with effusion lead to

## SEMILUNAR CARTILAGES, DISLOCATION OF

general laxity of the capsular and crucial ligaments, so that the joint becomes unstable, and secondary changes of chronic arthritis may follow, with hypertrophy of the synovial fringes or erosion of articular cartilage, progressing even to osteo-arthritis.

**Symptomatology.**—The patient's description of a typical injury is important. With the knee semiflexed and the foot fixed, the injury may be a blow on the outer side of the knee; with the foot fixed, there may be a twist of the body and thigh inwards; or with the body stationary the leg may be twisted outwards: the essential conditions are partial flexion of the knee and separation of the inner part of the articular surface of the femur from the corresponding surface of the tibia. At the moment of injury intense pain is caused, often described as "sickening." The cartilage then either slips back into its normal position or, by remaining displaced, causes locking of the joint; in the latter case the patient is unable to bear weight on the limb, and the knee cannot be fully extended. If the cartilage has slipped back into its place, or if reduction has been subsequently effected, there will be tenderness on pressure over the anterior end of the cartilage, just to the inner side of the ligamentum patellæ, and pain along the lower attachment of the internal lateral ligament elicited by abducting the leg at the knee-joint; and an abnormal degree of abduction may be possible. After the attack the joint becomes swollen and remains so for about a fortnight.

If after the first injury complete repair is not effected, the patient is liable to recurrent attacks of locking, and the amount of trauma necessary to produce such attacks becomes progressively less. Or there may be recurrent attacks of effusion from strain of the scar tissue resulting from the original injury, or attacks of sharp pain from the thickened anterior attachment of the cartilage being nipped in full extension of the joint. A slight degree of effusion may persist between the attacks, and there may be a sense of instability of the knee due to fine adhesions or to laxity of ligaments, and associated with some degree of wasting of the extensor muscles of the thigh.

Dislocation of the external semilunar cartilage, uncommon because it is not attached to the external lateral ligament, gives rise to symptoms similar to those of injury of the internal cartilage, but pain is referred to the

front or back of the outer part of the joint, according to whether it is the anterior or posterior part of the cartilage that has been injured.

**Diagnosis.**—The history of a typical initial injury is important, and if it has been followed by locking with loss of full extension the condition does not remain in doubt.

*Sprain of the internal lateral ligament* gives tenderness along its attachments, especially posteriorly, and pain on forcible abduction of the leg; the injury generally occurs with the knee in extension, and there is no history of locking and no special tender spot related to the anterior end of the semilunar cartilage. *Loose bodies in the joint* cause attacks of locking followed by effusion; the condition does not date from a typical injury, the locking is very often momentary and is reduced by no one special manipulation, and there is often an account of the loose body being palpable in different parts of the joint. *Recurrent nipping of the retropatellar pad of fat* occurs most commonly in elderly women; it causes attacks of sharp pain produced by some sudden movement of full extension. The pad can be seen and felt to be thickened, and there is tenderness on both sides of the patellar ligament. In *intermittent hydrops* each attack comes on after an interval which is definite for the individual case, and the attacks are independent of injuries or strains.

**Prognosis.** In cases treated by sufficient rest after the first attack, or by operation before permanent changes have taken place in the general structures of the joint, the prognosis is good. When, however, the ligaments have become lax, and there are changes in the articular cartilages and thickened synovial fringes, a permanently weakened knee-joint must be expected.

**Treatment.**—Reduction of the dislocated cartilage must be effected as soon as possible. Soon after the initial injury this can be done, as a rule, without an anæsthetic, but gas may be necessary in difficult patients. With the patient lying on his back, the thigh is fully flexed on the body and the leg is flexed on the thigh; the surgeon then presses the thigh outwards, inverts the foot, and suddenly pulls the knee straight. Sir Robert Jones, at the moment that he pulls, gets the patient to assist by suddenly extending his limb with a kick; if reduction is effected the patient is aware of it, and the knee can be brought to full extension. A bandage is then firmly applied to

## SEMILUNAR CARTILAGES

the knee over a thick layer of wool, and the limb is fixed on a straight patellar splint; this is kept on for ten days, but the patient is allowed to walk a little after five days. On the tenth day the splint may be discarded and the patient allowed to get about with wool and bandage still on, but he must avoid any sudden flexion.

Massage of the thigh muscles is begun on the third day, and small passive and active movements are practised from the tenth day; these are gradually increased so that the full degree of flexion is reached in about three weeks, without recurrence of effusion. The patient is then allowed to resume normal activity, the inner side of the heel of his boot being thickened so as to prevent strain on the internal lateral ligament; this should be kept up for two months, and in cases treated carefully from the beginning no further trouble is to be expected. If reduction cannot be effected, even with an anæsthetic, open operation is usually necessary.

In cases where reduction has not been carried out in the first instance there is slight loss of full extension, and passive attempts to procure this movement cause pain localized to the anterior end of the cartilage. In this late stage reduction can often be effected under anæsthesia by manipulations similar to those described for immediate reduction; otherwise, an open operation must be performed.

Operation is indicated also for cases which have reached the stage of recurrent dislocation with attacks of locking and effusion.

After operation no weight should be put on the limb for ten days: massage is then instituted, and gradually increasing active movements are allowed, reaching their full range usually in three weeks. Complete recovery is indicated by the thigh muscles regaining their full girth.

If, in cases of recurrent dislocation, operation is for any reason contraindicated, a cage-support, reaching from the upper third of the thigh to the lower third of the leg and allowing full extension and flexion but preventing rotation, should be employed; the inner side of the heel of the boot is thickened and the patient is trained to walk with the toes turned a little inwards, so as to transmit his weight through the outer part of the limb.

Severe injuries of a lateral sliding nature may cause simultaneous injury of both semilunar cartilages, by squeezing them out of place.

## SEMINAL STAINS

After such injuries prolonged rest and splinting are necessary, with treatment by massage and movements similar to that described above. Open operation is usually indicated.

C. W. GORDON BRYAN.

**SEMINAL STAINS.** — Examination with the naked eye and hand-lens, and chemical tests, are of much less importance in seminal than in blood-stains (see BLOOD-STAINS, EXAMINATION OF), and the only indisputable evidence of semen is that derived from microscopic examination. The dried stain stiffens cloth fabrics, and on light-coloured materials the colour is greyish yellow. When treated with a few drops of hot water the stains are said to become more yellow and to evolve a characteristic seminal odour. The chemical tests which have been proposed are not sufficient to prove the presence of seminal fluid. Microscopical examination aims at discovering at least one *unbroken* spermatozoon. The stain is cut out and placed in a watch-glass and moistened with a little distilled water, or distilled water rendered faintly acid with acetic acid. The watch-glass is covered to prevent evaporation, and after several hours the fabric is picked up with flat-pointed forceps and several glass slides are dotted with the adherent solution. The fluid in the meshes of the fabric is then squeezed out with the forceps and several other slides are prepared. The films are next fixed by being passed to and fro well above the Bunsen flame, and are afterwards stained. I have found carbol-fuchsin the best stain for routine work. The film is covered with stain, which is immediately poured off and the slide washed with a gentle stream of distilled water. After drying (either in air or high above the flame), the slide is examined with a  $\frac{1}{4}$ -in. objective, and any spermatozoa found are examined with the oil-immersion lens. For differential staining eosin and methyl-green may be used, the basal portion of the head being stained green, and the anterior part of the head and the long tail red. The average measurements of spermatozoa are—length 50-55  $\mu$ , head 4.3-5.2  $\mu$  long and 2.9-3.6  $\mu$  wide at its broadest part. Spermatozoa are so characteristic in appearance that complete specimens cannot be confused with any other thing. *Trichomonas vaginalis* is quite a different-looking object which should never be mistaken for a spermatozoon.

The more recent the stain, the more easily will spermatozoa be detected, other things

## SEPTICÆMIA AND PYÆMIA

being equal. Though they are fragile and disintegrate easily, they have been found in stains many years old; Roussin detected them after the long period of eighteen years. Perfectly dry stains on clean garments will usually yield better results than stains on garments soiled by urine and fæces; in the latter case putrefactive changes lead to their rapid disappearance.

A. ALLISON.

**SENILE DEMENTIA** (*see* DEMENTIA, SENILE).

**SENILE KERATOSIS** (*see* HYPERKERATOSIS).

**SENILE PARAPLEGIA** (*see* PARAPLEGIA, SENILE).

**SENILE WART** (*see* WARTS).

**SEPTIC MENINGITIS** (*see* MENINGITIS).

**SEPTICÆMIA AND PYÆMIA.**—Septicæmia is a condition in which pathogenic bacteria are present and living in the blood-stream; pyæmia, a condition of septicæmia in which the organisms are pyogenic and are producing metastatic abscesses in different parts of the body.

**Etiology and pathology.**—Bacteria are rarely introduced directly into the blood-stream. In every case of accidental infection, bacteria first become lodged in some local area, situated most commonly in the subcutaneous or submucous tissues. When the infection is at all severe, the products of bacterial metabolism, or toxins, pass into the blood-stream and produce symptoms characteristic of infective disease, such as rise of temperature, malaise, fascial pains in the limbs, and rise of pulse-rate. The *exotoxigenic* bacteria, of which the diphtheria bacillus is the commonest, may produce intense toxæmia, but rarely cause metastatic infection, though there may be a local spread of infection by continuity from the original site. The *endotoxigenic* bacteria differ from the exotoxigenic chiefly in that their toxins cannot be obtained artificially without destruction of the bacteria; within the body, however, there can be little doubt that the toxins do enter the circulation and produce the effects mentioned above. In the great majority of infections this local lesion and diffuse toxæmia represent the whole of the pathology; but in severe cases the bacteria may escape into the blood-stream and produce a septicæmia or pyæmia according to their

character. In every case in which metastatic infection occurs—for instance, when a patient with phthisis develops tuberculosis of the skin or bone—infection has been carried by the blood-stream, and a septicæmia has existed, at any rate for the time being. But when the diagnosis of septicæmia is made, a well-established condition is meant, in which the bacteria can actually be recovered from the patient's blood.

It is not possible to differentiate bacteria into septicæmic and non-septicæmic; any organism which is capable of inducing infection may, in favourable circumstances, escape into the blood-stream and produce a septicæmia. For instance, in pneumonia, which is primarily an infection of the lung, secondary infections of joints or meninges occasionally occur, and at the height of the fever pneumococci can often be recovered from the blood-stream. The same is true of the typhoid bacillus in the case of enteric fever. Both of these are grave and long-continued fevers, and suggest septicæmia. Gonococcal infections are, in the overwhelming majority of cases, local only, but here again joints are occasionally involved, and a few cases of general systemic infection with multiple lesions and endocarditis are to be found. The staphylococcus, too, produces chiefly local lesions, and though these are multiple, they are generally regarded as due to reduced resistance leading to ready infection from the skin where the staphylococcus is widely distributed, but occasional grave cases are to be found which yield positive blood-cultures. The same is true of the streptococcus, which is, however, much more likely to induce septicæmia than either the staphylococcus or the gonococcus. In general experience, more positive blood-cultures yield a streptococcus than any other organism. It will thus be seen that septicæmia is a matter of individual resistance on the part of the patient and individual virulence on the part of the organism, so that it is more correct to say that septicæmia is an accident which may occur in any infection, than that it is a peculiar state due to the infection by particular kinds of organisms. It should be remembered that the effects produced in the course of septicæmia are due to the toxins elaborated by the organisms, so that although they are likely to be more intense in degree, they are similar in kind to those produced by local infection.

Pyæmia cannot exist without septicæmia,



## SEPTICÆMIA AND PYÆMIA

nor septicæmia without toxæmia, and since it is the toxæmia which kills, the post-mortem appearances are those of poisoned tissues.

After death, in longstanding cases, there is marked wasting of the tissues, and decomposition sets in early. The blood remains fluid and accumulates in dependent parts, which are consequently discoloured. The viscera are in a state of cloudy swelling, and small hæmorrhages are often present in them and on serous surfaces. In septicæmia a site of infection can generally be found, in most cases communicating with some mucous surface. Examples are pneumonia or phthisis in the lungs, typhoid or other ulcers in the intestine, gonococcal infection of the genito-urinary tract, or traumatic lesions of the skin.

Pyæmic lesions vary with the site of the infection and the infecting organism. Abscesses may occur in the lungs, kidneys, spleen, brain, heart, or voluntary muscles. Any site exposed to physical injury is liable to attack. Streptococcal infections often produce endocarditis, and on the ulcers of the endocardium thrombi form and, becoming detached, may carry the infection to all parts; the lung, the brain, the spleen and the heart being the sites of election. Gonococcal infections show a preference for joints, tuberculosis for bones and serous and synovial membranes. When the intestines are involved, secondary abscesses may be found in the liver (portal pyæmia).

**Symptomatology.**—The symptoms are general and local. The general symptoms, being those of toxæmia, vary with the intensity of the poison and the powers of resistance of the patient. They include pyrexia, which differs according to the infecting organism, many infections being capable of diagnosis on the temperature chart alone. Characteristic instances are the evening rise and morning fall of tuberculosis, the abrupt rise and termination by crisis of pneumonia, the “steppage ascent,” the maintained fastigium, and the fall by lysis of typhoid. Rigors are common, and drenching sweats may occur.

**Circulatory system.** The pulse is usually rapid; where resistance is good it is full and bounding; in severe cases, small and thready. Mackenzie states that the rate increases from 8 to 10 beats with each degree Fahrenheit that the temperature rises above normal. In late stages the heart may dilate, as shown by an increased area of dullness and the development of mitral murmurs. If ulcerative endocarditis

be present, emboli are frequent. Embolism is usually accompanied by pain referred to the neighbourhood of the embolus. If in the lung, an area of dullness, with increased breath-sounds and sometimes a pleural rub, is to be found and hæmoptysis may occur; in the spleen, there are enlargement and tenderness; an embolus in the heart leads to sudden death, and one in the brain to partial or complete unconsciousness, often followed by some degree of paralysis, which, owing to the frequent involvement of the middle cerebral artery, frequently amounts to hemiplegia. An embolus in the kidney causes hæmaturia.

**Respiratory system.**—Respirations are increased in proportion to the pulse-rate, numbering about 24 with a pulse of 120. The usual signs of pneumonia, pulmonary embolism, or basal congestion from heart failure may be encountered.

**Alimentary system.**—Appetite is bad, thirst is great, the tongue as a rule is coated and sometimes raw, sordes may be present, vomiting and either constipation or diarrhœa occur; distension of the intestines is not uncommon. Special signs will be found if the primary source is in the tract.

**Nervous system.**—Headache is the rule, irritability and restlessness are common, and obscure pains are felt in the fascial structures and referred to the back and limbs. Light-headedness and actual delirium may occur, especially in grave cases. There is sometimes a terminal coma.

**Excretory system.**—The urine is diminished in amount; it is high-coloured and of high specific gravity, and often contains albumin in small quantities, and occasionally casts. The presence of the toxins may induce actual nephritis. The kidneys are not uncommonly sites of metastatic abscesses, and the occurrence of emboli with hæmaturia has been mentioned.

**Diagnosis.**—The existence of bacterial toxæmia is denoted by the presence of the febrile symptoms described; its nature is determined by the symptoms and physical signs detected, whether those of pneumonia, typhoid fever, meningitis, or other lesion. Pyæmia may be diagnosed when definite metastatic infections, such as emboli, abscesses, or arthritides, can be found. The chief difficulty arises in the distinction between septicæmia and toxæmia. Septicæmia is the graver condition, and when the temperature exceeds 102° F. for a period of days the probabilities are in favour of the

## SEPTICÆMIA AND PYÆMIA

presence of organisms in the blood. The diagnosis can only be made with certainty by the cultivation of bacteria from the blood. The usual method is to withdraw about 10 c.c. of blood from a vein with a sterile syringe, and to distribute this through half a dozen test-tubes, each containing about 10 c.c. of broth. (*See BACTERIOLOGY AND PATHOLOGY, CLINICAL*) Special media are used when particular organisms are suspected. The appearance of bacteria in these cultures in the hands of capable bacteriologists is diagnostic of infection. The organisms most commonly cultivated are the streptococcus and staphylococcus; the pneumococcus, influenza bacillus, gonococcus, and typhoid bacillus, together with others of the *B. coli* group, are also found occasionally. A point of importance is this: if a severe local lesion be present a septicæmia often follows, due to a streptococcus or other organism, while the causative organism may not itself be found in the blood. In such a case a secondary infection has developed in the course of an attack by a particular pathogenic organism. Such a secondary infection may occur in diphtheria. Some observers claim to be able to cultivate, or at any rate to isolate, the tubercle bacillus from the blood, but the experiment is extremely difficult, and has not received general confirmation.

**Prognosis.** Septicæmia and pyæmia are certainly grave conditions, the gravity varying with the degree of persistent septicæmia. By this it is meant that a pyæmia, a condition in which metastatic foci occur at intervals, and which may be due only to a transient septicæmia, is, in itself, not so grave a condition as one in which the bacteria are so numerous in the blood that a sample of 10 c.c. withdrawn from a vein at random contains enough bacteria to impregnate half a dozen tubes.

The existence of septicæmia indicates that the defensive arrangements of the body, which aim at preventing the escape of organisms from local lesions into the blood-stream, have broken down. This leads to wide distribution of the toxins, and, in the case of pyæmia, to an increase in the number of definite foci where they may be produced. Recovery, however, takes place in many cases, and more frequently than is generally realized. The prognosis varies a good deal with the nature of the organism and the site of the principal lesions. In practice, septicæmia is most frequently diagnosed by blood-culture in obscure cases where lesions such as pneumonic consolidation or typhoid

ulceration cannot be discovered by physical methods. The patient has perhaps no localizing signs, but is obviously ill and suffering from a high temperature and the general signs of fever, and blood-culture yields streptococci, the commonest septicæmic organism in such cases. Septicæmias of this kind are undoubtedly very grave, and a fatal issue is common. If a septicæmic infection proceed from an ulcerative endocarditis, so that the infection of the blood-stream is constant and infected coagula are readily detached from the ulcers and disseminated throughout the body as emboli, the prognosis is evidently almost as bad as it can be. Horder states that such cases never recover, and instances to the contrary are certainly excessively rare. Such a prognosis, however, is appropriate rather to an ulcerative endocarditis than to septicæmia in general. It should be borne in mind that septicæmia frequently occurs in pneumonia and that it is said to be demonstrable in four-fifths of all cases of typhoid fever, in both of which recovery is the rule, although the mortality is high. Consequently the antibacterial defences of the body are not put out of action in septicæmia, although they undoubtedly work at a disadvantage. The prognosis in septicæmia is, therefore, best estimated according to the clinical disorder which it accompanies. In typhoid and pneumonia the prognosis is that of a severe case of either infection; in ulcerative endocarditis it is exceedingly bad, and almost hopeless. In what may be called "accidental" septicæmias it is undoubtedly grave, but recovery is not impossible. In pyæmia, in which septicæmia is not demonstrable—that is to say, in cases of infection in which metastatic lesions occasionally occur, as in gonococcal arthritis or tuberculosis—the prognosis for life is fairly good, but there is often much destruction at the sites of the metastases, leading to suppuration or sclerosis, and producing results which vary with the site affected. The infection of a joint, for instance, is rarely followed by complete recovery.

**Treatment.**—The treatment of infections of any kind may be either specific or non-specific. The whole question of recovery from bacterial infection depends upon the power of the body tissues to elaborate antibodies to the invading bacteria. If it be decided not to use specific methods in any given case, the only thing to be done is to put the patient under the best conditions as regards food, air, and rest, so that he may have every chance of developing

## SEPTICÆMIA AND PYÆMIA

the necessary antibodies for himself, and to treat symptoms arising from the local lesions as they occur. If, on the other hand, the attempt is made to deal specifically with the infecting organism, it can be done in one of two ways, by attempting to induce either passive or active immunity (*see* IMMUNITY). At various times numerous anti-serums have been prepared with a view to *passive* immunity; their greatest success has been in diphtheria, which is primarily a toxæmia, and not a septicæmia. Diphtheria antitoxin contains antibodies to the products of the diphtheria bacillus and not to that organism itself. Its success depends on the fact that horses, which are the animals used for the production of antitoxin, have extraordinary powers of forming it, so that a serum can be produced which contains antitoxin in highly concentrated form. It is by no means proved that the bactericidal serums—those aimed at the streptococcus, gonococcus, typhoid bacillus and others—contain bactericidal substances in anything like the same proportion. It is, in fact, quite certain that they do not, hence the brilliant results obtained by the use of diphtheria antitoxin have not been obtained by the use of serums in septicæmias.

The alternative method is to try to induce *active* immunity, that is, to stimulate the tissues of the patient to produce their own antibodies. For this purpose vaccines, which are suspensions of bacteria themselves, killed by heat, are injected under the skin into healthy parts of the body. The hypothesis is that these killed organisms stimulate the tissues in which they lie to produce antibodies to them which pass into the circulation and become available against the bacteria causing the infection. It is assumed that the tissues at the sites of the actual infection have been damaged by the toxins, and so have failed to exercise their function of producing antibodies and to prevent the spread of the disease. The curative use of vaccines was first introduced by Wright in cases of localized infection only, and these are undoubtedly the cases in which the results are most satisfactory. How far it is wise to use vaccines in the septicæmias is a debatable point. In such diseases as pneumonia, in which the infection follows a short course and generally ends in recovery, many observers hold that it is wisest to leave well alone. Cases, however, treated by Willecox and Parry Morgan indicate that in their hands there is distinct advantage on the side of inoculation.

In cases of obscure septicæmia discovered by blood-culture, where, as has been said, the prognosis is bad, vaccines should always be used.

The antibodies produced as the result of accidental infection or deliberate inoculation are specific in character, consequently no good results are to be anticipated unless an accurate bacteriological diagnosis is made.\* This may be done by obtaining organisms from the blood, which is the best method, by obtaining them from some local site and assuming that they are causing septicæmia, or by testing the reactions of the blood to some of the known organisms, by a method such as the Widal reaction in typhoid fever. A diagnosis having been made, the treatment will depend on the gravity of the case. Only a few bactericidal serums are available, and if a special serum be required in any given case it takes a long time and is very expensive to prepare, whereas a vaccine can, as a rule, be made in a few days. In a case of moderate severity, when there is no immediate urgency, a vaccine should be prepared from the most promising site and injections undertaken.

In a streptococcal infection, which is that most commonly requiring treatment, a dose of one million organisms should be given, and be followed by  $2\frac{1}{2}$  millions in two days; this may either be repeated or doubled in three days more, according to the clinical indications. The doses and the intervals may be gradually increased until 25 millions are given every week or ten days. It is extremely important that the initial dosage in a septicæmia should be very small. In very grave conditions, such as puerperal septicæmia, if streptococci be found to be numerous in the uterine discharge, a serum may be used. There is no doubt that many desperate cases of this kind have recovered under this treatment. A polyvalent serum should be used, that is, one prepared by using many different strains of streptococci, and in doses of 20 to 30 c.c., to be repeated if necessary in twelve hours. It may be that such serums contain antitoxins and so produce immediate relief by neutralizing the toxins in the circulation, or it may be that they also act, in part, as vaccines, if the animals yielding the serum are in a state of bacteriæmia. Excellent results are occasionally obtained, although there are undoubtedly many failures.

\* Recent work has thrown some doubt on the purely specific nature of antibody-formation.

## SEROLOGICAL DIAGNOSIS

During inoculation-therapy the case must be treated as one of severe illness. In the early stages the patient must be kept strictly in bed, with open windows, or preferably out of doors if that be possible. Meals should be small and frequent, thirst must be gratified. In a chronic septicæmia, patients, though obviously very ill, sometimes do not feel so, and express a desire to get up or go about. There seems to be no objection to allowing this course, so long as the patient obtains pleasure from it. One of the most striking cases of recovery in my experience was that of a lady who insisted on going out for drives when suffering from grave streptococcal septicæmia with rigors and emboli.

D. W. CARMALT-JONES.

**SEROLOGICAL DIAGNOSIS.**—Infection or artificial immunization\* leads to the development in the blood-serum of antibodies, which react in a characteristic manner with the antigen (e.g. the infecting organism) both *in vivo* and *in vitro*—immunity or antibody reactions. These reactions exhibit a high degree of specificity; thus, the immune serum acts only with the antigen which is responsible for the immune state, and fails to react with another antigen not of the same biological character as that used for immunization. In this way highly specific and extremely delicate tests can be applied in the diagnosis of infective disease. Thus, (1) in a suspected infection the serum can be tested for the presence of antibodies to the particular organism known to be the causal agent of the disease in question; (2) a bacterial species or type can be recognized or identified by means of a specific antiserum which has been obtained by immunizing an animal with dead cultures of the same variety; (3) in a particular infection the fact that the patient's serum contains specific antibodies to an organism which has been isolated from the case is strong evidence that this organism is pathogenic. Various antibody reactions have been employed in this way, e.g. agglutination, precipitation, complement deviation, bacteriolysis, opsonic action. Thus, the agglutination of *B. typhosus* and the paratyphoid bacilli by the serum of suspected enterica cases (Widal reaction) has proved of the greatest practical value in the diagnosis of these infections, and the Wassermann reaction (complement deviation), though not a true immunity reaction,

\* The principles of immunity, on which sero-diagnostic methods depend, are expounded under the head of IMMUNITY.

now constitutes a routine test in the diagnosis of syphilis. In bacteriological laboratory work the accurate identification of various pathogenic organisms can only be established by means of known specific antisera which react with the homologous organisms, e.g. *B. typhosus*, *B. paratyphosus-A* and *-B*, *V. cholerae*, etc. Antibody reactions (precipitation, complement deviation) can also be utilized for detecting the presence of minute amounts of human blood as in medico-legal investigations.

In the course of an infection the individual may develop a specific hypersensitiveness to the products of the infecting organism when introduced into the tissues (allergy); a local inflammatory reaction is produced at the site of injection, and can be readily observed, e.g. after intradermal injection. The tuberculin and luetin (syphilis) reactions are of this nature.

The extent to which serum tests are utilized in diagnosis depends on the antibody development during the illness and on the other diagnostic methods available; in a short acute disease like cholera, where antibodies only appear in the serum in convalescence, sero-diagnostic tests cannot be of much practical value; in cerebro-spinal meningitis, also, though antibodies appear in the serum, and a serum test could be employed for diagnosis, the detection of the causative organism represents the most certain method of diagnosis. In enterica illness, however, unless the case comes under observation in the early stages, when blood-culture is likely to yield positive results, the agglutination test may provide the only means of confirming the clinical diagnosis, for the isolation of the causative organism from the excreta is uncertain and involves a considerable amount of detailed laboratory work.

Agglutination tests are in general less complicated as regards technique than other immunity reactions, and, where specific agglutinins have been shown to be characteristic of the immune serum, this reaction is usually selected in preference to other immunity tests (e.g. complement deviation, bacteriolysis).

### AGGLUTINATION REACTIONS

**Enterica infections. Widal reaction.**—The test has been extensively used as a routine laboratory method, and an analogous reaction has been applied in the diagnosis of paratyphoid infections (*B. paratyphosus-A* and *-B*). It is, however, somewhat restricted in its practical value. Agglutinins may be late in

## SEROLOGICAL DIAGNOSIS

developing, or may not develop to any appreciable degree during the whole of the illness; hence a negative reaction is of little significance. Preventive typhoid or paratyphoid inoculation, which *per se* leads to the production of specific agglutinins, considerably limits the value of the test in cases developing enterica subsequently to inoculation. Agglutinins do not usually appear until the seventh day; thus the test is hardly applicable in the earliest stage of the disease. Normal serum may agglutinate typhoid and paratyphoid bacilli in low dilutions, and certain strains of paratyphoid bacilli may be spontaneously agglutinable in salt solution without serum.

In the uninoculated person the presence in the serum in increased amount of agglutinins for *B. typhosus*, *B. paratyphosus-A* or *-B*, is strong evidence of infection with the particular organism. Additional confirmation is established if, during the illness, the agglutinin content shows a considerable increase on repeated testing and remains high in the early stages of convalescence.

It is important to remember that different strains of these organisms vary as regards agglutinability, and also differ in agglutinability at different times. It is necessary, therefore, that strains should be selected for the test whose behaviour with a number of normal serums and an immune serum has been previously ascertained.

It is also essential that agglutination tests should always be carried out by a strictly quantitative method.

### Method of carrying out agglutination tests.

**Collection of blood specimens.**—The lobe of the ear or the finger at the root of the nail is sterilized with spirit, and a puncture is made by a deep thrust of a sterile Hagedorn needle; when a large drop of blood has exuded, the end of the bent limb of a Wright's blood capsule is dipped into it and blood passes into the capsule by capillary action. The capsule is filled three-quarters full. If blood does not flow freely, pressure may be applied by winding a narrow bandage round the proximal phalanx. To close the capsule the free end is heated and the tip sealed in a flame. As this end cools, the blood is retracted from the bent limb of the capsule, and the other end may then be sealed. The serum is separated by centrifugalization, and on opening the capsule can be pipetted off.

**Bacillary emulsion.**—Subcultures of suitable strains of *B. typhosus*, *B. paratyphosus-A* and

*-B*, are made on agar slopes and incubated for eighteen hours at 37° C. These cultures are emulsified in 3–5 c.c. of 0.85 per cent. sodium chloride solution so as to produce a turbid emulsion. Agglutination occurs with dead cultures, and some serological workers prefer to use bacillary emulsions made up in formalized salt solution (0.1 per cent. formalin) Standardized suspensions (Dreyer)—formalinized bouillon cultures whose agglutinability has been standardized by comparison with previously prepared standards—have been used with a view to obtaining strictly comparable results over a large number of tests.

**Serum dilutions.**—The serum is made up in a series of dilutions with normal saline in small test-tubes so as to represent the following concentrations, viz. 1 : 25, 1 : 50, 1 : 100, 1 : 200, 1 : 400, 1 : 800, 1 : 1,600\*—and a certain fixed amount (e.g. 0.3 or 0.4 c.c.) of these dilutions is mixed with equal volumes of the three emulsions. In this way each of the three organisms is tested with the following series of serum dilutions, viz. 1 : 50, 1 : 100, 1 : 200, 1 : 400, 1 : 800, 1 : 1,600, 1 : 3,200. To each series should also be added a control test containing bacillary emulsion but no serum.

**Macroscopic or sedimentation method.**—The various mixtures are transferred to narrow tubes 3 by  $\frac{1}{8}$  in.; the tubes are placed at 37° C. for an hour and a half, and then allowed to stand at room temperature for half an hour; if agglutination occurs, the clumps or flocculi can be observed with the naked eye. In the course of time the clumps sediment in the bottom of the tube, leaving the supernatant fluid clear; after twenty-four hours, final readings can be made by observing the amount of sediment in the tubes.

With Dreyer's emulsions the tubes should be placed at 50°–55° C. for two hours, and then at room temperature for fifteen minutes, before readings are made.

**Microscopic method.**—The effect can also be observed by examining microscopically hanging-drop preparations from the various mixtures.

**Criteria of a positive reaction.**—Marked agglutination of the respective organisms in the following dilutions is significant in early cases not previously inoculated : *B. typhosus*,

\* For measuring small quantities of fluid (e.g. serum, salt) and preparing dilutions, etc., graduated glass pipettes are most suitable. Two pipettes are required—(1) total capacity 1 c.c., graduated to 0.025 c.c.; (2) total capacity 0.1 c.c., graduated to 0.0025 c.c.

## SEROLOGICAL DIAGNOSIS

1:100; *B. paratyphosus-A*, 1:50; *B. paratyphosus-B*, 1:200. In the later stages of the disease, however, higher dilutions may produce marked agglutination, and the higher the dilution in which agglutination occurs the more conclusive is the test.

In the case of *B. paratyphosus-A* infections, the development of agglutinins may be slight even in the later stages of the disease, and agglutination of this organism by a 1:20 dilution has been frequently accepted as evidence of the infection.

In the microscopic method marked agglutination of *B. typhosus* by a dilution of 1:30 after half an hour has usually been regarded as a positive result.

The necessity for a strictly quantitative method is undoubted; the higher the dilution in which agglutination occurs the more conclusive is the result as evidence of a specific reaction to the particular organism. An infection with one organism of the group may lead to some degree of group reaction (coagglutination) in addition to the specific effect; and though in the enterica group coagglutination effects are usually slight, a quantitative determination is necessary to show with which organism agglutination is most distinct. When only feeble agglutinating effects are demonstrated, the test should be repeated after a few days; in this way more conclusive proof may be elicited.

**Agglutination tests in persons inoculated with typhoid vaccine.**—After inoculation with a *B. typhosus* vaccine, postinoculation agglutinins for this organism may persist in the serum for long periods. Agglutination of *B. typhosus* by a serum dilution of 1:1,000 after six months have elapsed from the time of inoculation is, however, strongly suggestive of a typhoid infection. If the interval is less than six months, little reliance can be placed on the test, and even in undoubted typhoid infections, proved by isolation of the causative organism, the reaction may not reach a titre of 1:1,000. Hence in typhoid-inoculated cases the test has a very restricted practical value in the diagnosis of *B. typhosus* infections.

In paratyphoid infections a previous typhoid inoculation does not materially affect the value of the test, provided a quantitative estimation is made and the results are correctly interpreted; both *B. paratyphosus-A* and *-B* infections may produce a marked reaction to the specific organism, reaching high titres, e.g. 1:2,000 and 1:4,000; but a paratyphoid infection also produces an increase in the

typhoid agglutinins which may act in as high titres as the paratyphoid agglutinins, though as a general rule the end-titre for *B. typhosus* is lower than that for the paratyphoid bacillus.

In a case inoculated with typhoid vaccine and subsequently developing an enterica illness the occurrence of high-titre agglutinins to one of the paratyphoid bacilli, even though associated with an equally pronounced agglutination effect on *B. typhosus*, is strong evidence of the particular paratyphoid infection.

**Agglutination tests in persons inoculated with typhoid-paratyphoid vaccine.**—A considerable amount of detailed work has been carried out with a view to eliciting diagnostic proof of enterica infections by charting the curves of agglutinin content in the serum during the disease; the interpretation of these curves is still a matter of controversy, and it may be said generally that in triply-inoculated persons agglutination tests are of little practical value compared with methods which aim at direct isolation of the causative organism.

For the detection of typhoid carriers the agglutination reaction provides a useful preliminary test; but proved carriers may show no reaction, so that the test can hardly be substituted for the bacteriological examination of the excreta. Mild enteric cases may often escape notice owing to the absence of distinct clinical symptoms, and these cases are a considerable source of danger in disseminating infection. In such persons the Widal reaction may be definitely positive, and is of value in their detection.

It is to be noted that a positive Widal reaction has been recorded in cases other than enterica, e.g. in cases of typhus fever.

**Food poisoning.**—In *B. enteritidis* (Gärtner) infections, specific agglutinins appear in the serum, and, in the absence of direct evidence by isolation of the organism, the agglutination reaction can be used for diagnosis.

**Bacillary dysentery.**—Agglutination phenomena in dysenteric infections are variable; they may be absent and are rarely so marked as in typhoid and paratyphoid cases. For diagnosis it is necessary to test the serum with each of the different varieties of classical *B. dysenteriae*-Shiga and the five serological types of the Flexner-Y group (Andrewes and Inman, Murray); the test is thus too laborious for routine work, and in addition to the classical varieties a number of atypical strains may be responsible for acute dysentery (Machle). Normal serum may agglutinate dysentery

## SEROLOGICAL DIAGNOSIS

bacilli in low dilutions. Agglutination tests are therefore of little practical value, and if cases come under early observation the isolation of the causative organism is the most certain method of diagnosis.

**Undulant fever** (*M. melitensis* and *paramelitensis*).—In these infections the serum may agglutinate the specific organism as early as the fifth day; it is advisable to heat the serum at 56° C. for half an hour to eliminate non-specific agglutination by fresh normal serum.

In **cholera**, agglutinins only develop in convalescence, and the agglutination test has no application in diagnosis during the acute stage. It may be of value in deciding whether a convalescent has actually suffered from a *V. cholerae* infection.

In **plague** also specific agglutinins occur in the blood at too late a period to be of value in diagnosis. The reaction may be completely absent even in convalescence.

In **tuberculosis** the agglutination reaction has been used, but the results are conflicting and the test has little or no practical value.

Agglutinins for *Spirillum pallidum* have been described in the serum in **syphilis**, but the diagnostic use of the test has not yet been established.

The **Weil-Felix reaction** is the agglutination of an organism of *B. proteus* type by the serum of **typhus fever** cases, and has been found of some value in diagnosis. It may be regarded as a heterogenetic reaction characteristic of this disease.

**Conglutination** (Bordet and Gay, Streng).—This term has been applied to the increased agglutinating effect produced by heated ox-serum acting along with a specific antiserum and complement. It has been claimed that the agglutinating effect is intensified and is more specific.

**Identification of organisms by agglutination tests.**—Immune animal serums are largely used in bacteriological routine for the final identification of certain species or types of bacteria after preliminary classification by cultural tests. Certain pathogenic organisms so closely resemble one another in biological characters that their identification depends entirely on serological methods (e.g. *B. paratyphosus*-*B* and *B. Gärtner*), and various species can be subdivided further into different types by serological tests. These reactions are highly specific.

**Hæmagglutination.**—Just as specific agglutinins are developed in the serum of an animal

immunized with bacteria, so also hæmagglutinins appear in the serum of an animal injected with red corpuscles, and produce clumping of the homologous red cells in suspension or in the blood *in vivo*. Apart from immunization, normal serum may contain agglutinins to the corpuscles of other animals and even to those of the same species (isohæmagglutinins). Since the serums of certain individuals may agglutinate the corpuscles of others, in carrying out blood transfusions intravascular agglutination might occur, with serious results to the recipient. It has been found that human blood can be classified in respect to isoagglutinins into four groups (Moss):—

I. Serum does not agglutinate corpuscles of the other groups; corpuscles agglutinated by serums of groups II, III, and IV.

II. Serum agglutinates corpuscles of groups I and III; corpuscles agglutinated by serums of groups III and IV.

III. Serum agglutinates corpuscles of groups I and II; corpuscles agglutinated by serums of groups II and IV.

IV. Serum agglutinates corpuscles of groups I, II, and III; corpuscles not agglutinated by serums of other groups.

No serum agglutinates corpuscles of the same group. Isohæmolysis is closely related to isoagglutination.

In selecting a donor for transfusion, agglutination and hæmolytic tests should, if possible, be carried out beforehand to ascertain the effect of the donor's serum on the recipient's cells and—more important—the effect of the recipient's serum on the corpuscles of the donor. When specimens of blood from known groups II and III cases are available, any other specimen can be classified; in this way a donor belonging to the same group as the recipient can be selected. **Method:** 5 c.c. of blood are withdrawn by vein puncture from the case to be tested; 1 c.c. is added to a sodium citrate salt solution to prevent coagulation, and the mixture is centrifugalized to deposit the corpuscles, which are washed two or three times with salt solution; the serum is separated from the remaining 4 c.c. by centrifugalizing. The corpuscles are made up in a 10-per-cent. suspension with salt solution. The serum and corpuscles whose interaction is to be determined are mixed together in small test-tubes (4 vols. of serum to 1 vol. of corpuscular suspension) and incubated for two hours at 37° C., when the occurrence of agglutination or hæmolysis is noted. A simpler method is

## SEROLOGICAL DIAGNOSIS

to mix a small platinum loopful of the blood with a large drop of serum on a microscopic slide, and to examine under the microscope after several minutes.

### PRECIPITIN TESTS

**Bacterial precipitins.**—While specific bacterial precipitins, like agglutinins, may appear in the blood of patients suffering from various infections, their detection is rarely applied in diagnosis; precipitin reactions present more difficulties in technique and are less delicate than agglutination tests, so that the latter are usually selected for routine serological work. Thus, in the identification of organisms by serological methods, agglutination tests are generally preferred to precipitin reactions.

Precipitin reactions have been utilized for the detection in blood, urine, exudates, tissues, etc., of specific soluble substances derived from the causal organism, which yield a precipitate along with the homologous antiserum. In **pneumonia** soluble pneumococcus substances are present in the blood and urine at an early stage of the illness (e.g. during the first day), and can be demonstrated by the precipitin reaction with a specific antiserum for the respective type of pneumococcus. In **cerebro-spinal meningitis** similar soluble substances, derived from the meningococcus, may appear in the cerebro-spinal fluid and along with an antimeningococcus serum produce a precipitation reaction; the reaction has been utilized in diagnosis. It has been claimed that in **typhoid fever** specific precipitinogens are present in the serum at an early stage of the affection and can be recognized by the precipitin reaction with antityphoid serum. In the same way in **anthrax** the presence of *B. anthracis* in blood and tissues may be determined by Ascoli's thermoprecipitin test. The tissue or blood is boiled with 10 vols. of normal saline containing 0.1 per cent. of acetic acid. After cooling, the fluid is filtered; a certain amount is then added slowly to an anthrax-immune serum in a test-tube; a white ring (precipitate) forms at the line of the junction. An analogous reaction has been employed for the detection of material containing *B. pestis*, e.g. tissues of infected rats.

**Serum precipitins.**—When the serum of an animal is injected in repeated doses into another animal of different species, the serum of the latter develops a specific antibody which produces a precipitate when mixed with the serum of the former. In this way the presence of

the serum or blood of a particular animal species may be recognized if the homologous antiserum is available. The reaction is used for the detection of human blood-stains. (See BLOOD-STAINS, EXAMINATION OF.)

An analogous method has been applied for the detection of meat adulteration, e.g. the presence of horse or dog flesh in sausages. These reactions, however, are not so delicate as complement-deviation tests (*see below*).

### BACTERIOLYTIC TESTS

Bacteriolysins, like other antibodies, are highly specific for the homologous bacterium, and the bacteriolytic or bactericidal test may also be employed in the sero-diagnosis of various infections and for the accurate identification of certain organisms. Bactericidal tests have, however, little practical application.

The classical **Pfeiffer's reaction** (bacteriolysis of *V. cholerae* injected with an anticholera serum into the peritoneum of an animal) is commonly used in the serological identification of the cholera vibrio. The test may be carried out *in vitro*; the bacteriolysin is inactive without complement, and this is supplied by adding fresh guinea-pig's serum. The bacteriolytic reaction should always be carried out in the case of a vibrio which is not agglutinated or reacts in low titres with the anticholera serum, so as completely to exclude a *V. cholerae* infection.

### HÆMOLYTIC TESTS

A suspension of red corpuscles "sensitized" with a hæmolytic immune body is used in complement-deviation reactions as an indicator of complement action (*see IMMUNITY*). Ox or sheep corpuscles sensitized with an homologous immune rabbit serum are lysed (laked) by minute amounts of fresh guinea-pig's serum, e.g. 0.005 c.c. for 0.5 c.c. of 5-per-cent. suspension of blood, and this "hæmolytic system" is found best adapted for estimating complement absorption. The immune body is prepared by injecting a rabbit intravenously or intraperitoneally with increasing doses of the corpuscles washed free of serum. Powerful immune serums are obtained in this way (e.g. minimum hæmolytic dose for 1 c.c. suspension with excess of complement -0.001 c.c.). The corpuscles sensitized with this immune body are lysed by the complement. Neither immune body nor complement separately has any lytic effect. It is usual to sensitize with a considerable excess of immune serum (five minimum doses), as



## SEROLOGICAL DIAGNOSIS

the maximum effect of complement does not occur with the minimum dose of immune body.

**Serum diagnosis of paroxysmal hæmoglobinuria** (Eason-Donath-Landsteiner reaction).—Paroxysmal hæmoglobinuria is characterized by the presence in the serum of an autolysin, i.e. an immune body which, with complement, produces lysis of the individual's own corpuscles. The immune body, however, only combines with the cells at low temperatures. The complement which produces intravascular hæmolysis along with the immune body is active at 37° C. For the test, blood is withdrawn from a vein and part is added to citrate-salt solution and centrifugalized; the corpuscles are then washed with salt solution and suspended in saline. Part of the blood specimen is placed in a dry tube and centrifugalized to separate the serum. Corpuscles and serum are mixed in definite amounts, and the tubes are placed at 0° C. in ice for one hour and then incubated at 37° C. for two hours. The occurrence of hæmolysis denotes a positive result.

**Cobra venom hæmolysis.**—Cobra venom exerts a marked lytic action towards the red corpuscles of certain animal species (e.g. human corpuscles), and this effect may be greatly influenced by the presence of serum. According to Much and Holzmann, in cases of the Kraepelin type of dementia præcox and of depressive mania the blood-serum shows a marked inhibiting effect, and this reaction has been regarded by some as diagnostic of these conditions. Other investigators have not, however, confirmed these observations.

It has also been suggested that the power of the serum in cases of cancer to aid venom hæmolysis is of diagnostic significance, but the specificity and practical value of the test have not been established.

Considerable variations in the effect of human serum as regards its power to aid or inhibit venom hæmolysis may occur, but it is doubtful if these changes can be definitely associated with particular diseases

### COMPLEMENT-DEVIATION TESTS

One of the properties of an immune serum is the power of fixing complement in the presence of the homologous antigen. This reaction occurs with an organism and its homologous antiserum—**Bordet-Gengou reaction**. The deviation of complement is determined in the following way: The antigen is mixed with the

antiserum in appropriate amounts, and then complement—e.g. fresh guinea-pig's serum—is added; the mixture is incubated for about an hour and a quarter, and red corpuscles sensitized with an homologous immune body (hæmolytic system) are added. If hæmolysis occurs after a further period of incubation, complement is apparently unabsorbed and is free to act on the sensitized corpuscles. If no lysis occurs, absorption has taken place. This reaction constitutes an extremely delicate and specific test. Thus, an antigen—e.g. a particular bacterial species—can be recognized if the homologous antiserum is available, and in the same way the presence of specific antistances can be determined. It has therefore been extensively applied in diagnosis. By means of an homologous antiserum also, the blood or serum of a particular animal species can be identified. Complement-deviation methods, like agglutination reactions, have thrown additional light on the classification and grouping of various bacteria.

**The syphilis reaction of Wassermann, Neisser, and Brück.**—In sero-diagnostic routine this is the most commonly-employed complement-deviation test, and has proved of the utmost value in practical medicine as a specific method for the recognition of a syphilitic infection. Though it does not depend on the interaction of the infecting agent with a specific immune body, the test exemplifies complement-deviation methods generally.

It was originally found that a watery extract of the liver from a syphilitic foetus containing a large number of spirochaetes, along with the serum of marked cases of syphilis, possessed the power of annulling the action of hæmolytic complement. It was concluded that the effect depended on complement-deviating immune bodies in the serum which reacted with the specific organism or its products, as in the case of the Bordet-Gengou reaction. It was subsequently shown, however, that an alcoholic extract of normal animal tissues (e.g. liver, heart, etc.) acted as "antigen" with syphilitic serums in deviating complement; the active constituents of these extracts were ultimately shown to be lipoids (lecithin and cholesterin). The effect is, therefore, not a true immunity reaction, though so highly characteristic of syphilis as to constitute a specific test. The actual nature of the reaction is obscure; it is to be noted, however, that the difference between the syphilitic serum and normal serum is quantitative; the reaction therefore depends on

## SEROLOGICAL DIAGNOSIS

the power of a lipid emulsion to fix more complement in the presence of a syphilitic serum than with normal serums

**Method.**—The *blood specimen* is best obtained by vein puncture, as a considerable amount of serum may be required. A 10-c.c. syringe is sterilized in boiling water. The skin at the bend of the elbow is cleansed and sterilized, and the veins are rendered turgid by applying several turns of a bandage or a turn of rubber tubing round the arm about the middle of the biceps. The syringe needle is inserted into a prominent vein and 5–10 c.c. of blood are drawn into the syringe and then transferred to a sterile test-tube. After the blood has coagulated, the clot is freed from the side of the tube by shaking or rotation to enable the serum to separate.

**Antigen.**—An alcoholic extract is prepared from ox or sheep's heart or ox liver; 20 grm. of the minced tissue are ground up in a mortar with sand and extracted with 100 c.c. of alcohol. An alcoholic solution of the lecithin fraction of such an extract (the ether-soluble, acetone-insoluble portion) may be used. Cholesterin is added to the extract or the lecithin solution and increases its antigenic power (Browning, Cruickshank and MacKenzie). This antigen is made into an emulsion in salt solution (1:12 dilution).

Serum is heated at 55° C. for half an hour to inactivate the complement. Normal serum may also exert a non-specific deviating effect with the antigen; this is eliminated by heating.

The *complement* generally used is that of the guinea-pig (fresh guinea-pig's serum, eighteen hours after withdrawal of the blood).

**Hæmolytic system.**—Ox corpuscles washed free of serum with normal salt solution, and suspended as a 5-per-cent. suspension of blood in salt, are sensitized with a hæmolytic immune body (serum of a rabbit immunized with ox corpuscles).

The test should be carried out by a quantitative method so that the actual power of the antigen and serum to fix complement can be estimated.

A preliminary determination of the minimum hæmolytic dose (M.H.D.) of the complement for 0.5 c.c. of the sensitized blood is made. The M.H.D. usually lies between 0.005 and 0.01 c.c.

Fixed amounts of antigen emulsion (0.5 c.c.) and serum (0.05 c.c.) are mixed with varying quantities of complement—e.g. (1) 4 M.H.D., (2) 8 M.H.D., (3) 14 M.H.D.—in small test-

tubes and incubated for an hour and a quarter at 37° C. Control tests, in which the same amounts of antigen and serum are tested separately with complement, are included—e.g. with antigen, (1) 1 M.H.D., (2) 2 M.H.D., (3) 4 M.H.D., and with serum diluted with 0.5 c.c. salt solution, (1) 1 M.H.D. and (2) 2 M.H.D. The dose of complement is also re-estimated, the various amounts tested being added to 0.5 c.c. of saline and incubated with the other tubes for an hour and a quarter before blood is added.

After incubation 0.5 c.c. of the sensitized blood is added to each tube, and the tubes are again incubated for one hour.

The degree of hæmolysis in each tube is then noted. It is essential also that known negative and positive serums should be tested at the same time, as controls.

The occurrence of complete inhibition of lysis in the first tube (4 M.H.D.) may be regarded as a criterion of a positive reaction, provided that the negative control serum shows complete or almost complete lysis in this tube. Of course, in the majority of positive reactions the deviation is more marked and the result therefore more conclusive.

Positive reactions are obtained in 95 per cent. of cases of syphilis in the secondary stage; in 75 per cent. of tertiary cases; in 95 per cent. of congenital cases with active lesions, and in 50 per cent. of latent cases; in 70 per cent. of cases of locomotor ataxy, and in 96 per cent. of cases of general paralysis.

Among diseases common in Great Britain a positive reaction may be regarded as undoubted evidence of syphilis. In frambæsia, which is due to an allied organism (*Spironema pertense*), positive reactions are also characteristic; positive results have been recorded, too, in cases of malaria, leprosy, relapsing fever, and trypanosomiasis. It is to be remembered that in cases clinically non-syphilitic the occurrence of a positive reaction may depend on a latent concomitant infection with the virus of syphilis.

In the primary stage, the examination of the exudate of the chancre for the presence of spironemata should constitute the first laboratory test, for the Wassermann reaction may only be positive after two to four weeks from the earliest appearance of the chancre. The reaction may, however, be definitely positive after the sore has healed but before secondary symptoms occur, so that it is invaluable in

## SEROLOGICAL DIAGNOSIS

establishing the diagnosis in such cases with a view to immediate treatment.

A negative result in supposed tertiary cases is of less significance than in suspected secondary or congenital cases.

In cerebro-spinal syphilis the serum may react negatively while the *cerebro-spinal fluid* yields a positive result. A negative reaction with both serum and spinal fluid is significant in excluding a possible general paralysis.

In the differential diagnosis of tertiary syphilis and malignant tumours (e.g. of rectum, tongue) a positive reaction should not be accepted as definite evidence that the existing condition is a syphilitic lesion, e.g. gumma; a syphilitic infection may coexist with malignant disease, and the investigation of the case should be pursued further, e.g. histological examination of the tumour or ulcer.

Not only has the reaction proved of the greatest value in routine diagnostic work, but it has established the syphilitic origin of various conditions of doubtful etiology, e.g. aneurysm, aortic disease, primary optic atrophy, and paroxysmal hæmoglobinuria.

The Wassermann reaction is also of importance in deciding the advisability of marriage in the case of syphilitics; thus, in the early latent stages the patient may still be highly infective and the reaction may be the only means of diagnosis. A positive reaction in a person who has had the disease for many years does not necessarily signify infectivity.

**Provocative injection of salvarsan.**—Occasionally in latent syphilis with a negative reaction a dose of salvarsan may lead to the development of a positive reaction (commonly after a week), possibly by liberation of toxin from the killed organisms or by stimulation of the infection. This method is sometimes of value in detecting latent cases with otherwise negative reactions.

**The Wassermann reaction in controlling treatment.**—A positive reaction signifies the presence of an active lesion or generalized action of the parasite. The disappearance of the reaction during treatment may be accepted as a criterion of effective therapy. Also, after a negative reaction has been obtained as the result of preliminary treatment, the recurrence of a positive reaction later indicates the necessity for further treatment. In antisyphilitic therapy the maintenance of a negative reaction should be aimed at. During treatment the serum should be tested from time to time, and after a complete course of treatment

tests should be made at intervals for several years. If a provocative injection of salvarsan also fails to elicit a positive reaction, the result is suggestive of the efficacy of treatment.

In tertiary and congenital syphilis treatment has less effect in rendering the serum negative; in locomotor ataxy and general paralysis it has been claimed that salvarsan has little influence in producing a negatively reacting cerebro-spinal fluid, while in interstitial syphilis of the central nervous system this result usually follows thorough treatment.

**The Sachs-Georgi syphilis reaction.**—This reaction, which has been advocated as a substitute for the Wassermann test, depends on the occurrence of precipitation or flocculation in a mixture of syphilitic serum and cholesterolized alcoholic tissue extract (i.e. the antigen used in the Wassermann test). The precipitable substance is apparently the lipoids present in the antigen. There is a distinct parallelism between the Wassermann and the Sachs-Georgi reactions, but it is doubtful whether the flocculation test is sufficiently sensitive as compared with the Wassermann reaction. The Sachs-Georgi test is more easily carried out, but 18–21 hours may elapse before a definite result is obtained.

**Complement-deviation tests in the diagnosis of bacterial infections.**—Complement-deviation tests are not extensively used in routine diagnosis; the agglutination test, if applicable, provides a simpler method, in which the technique is less complex and attended with less chance of error. Group reactions, analogous to that noted in the case of a specific agglutinating serum, also add to the practical difficulty of applying these tests.

In **typhoid fever** specific complement-deviating antisubstances can be demonstrated in the serum, but the corresponding test has no advantages over the agglutination reaction and is more difficult to carry out.

In **gonococcus infections** the complement-deviation test has proved of considerable practical importance, especially in the diagnosis of chronic infections and the various complications of the disease which present difficulties in diagnosis by direct bacteriological methods. The reaction is highly specific, but a negative result does not exclude gonorrhœa, and the reaction is absent in the early stage of the disease. The test is especially useful in the diagnosis of gonorrhœal arthritis, iritis, pyosalpinx, and doubtful gynaecological conditions. It has been supposed that a negative

## SEROLOGICAL DIAGNOSIS

reaction after treatment is suggestive of "cure."

**Glanders.**—A complement-deviation test has been used in veterinary diagnosis and is regarded as more reliable than the corresponding agglutination test. This reaction has also been employed in the diagnosis of human infections.

**Tuberculosis.**—A complement-deviation reaction may be elicited with patient's serum and an antigen prepared from living cultures of the tubercle bacillus (an emulsion in salt solution or a bacillary extract). The fact that some normal serums and frequently syphilitic serums react with the tubercle antigen (non-specific reaction) has detracted from the practical value of the test. According to Fraser the reaction is positive in 42.3 per cent. of tuberculous cases, and negative in 96.6 per cent. of apparently normal subjects. Besredka has used an antigen prepared from tubercle bacilli grown in egg-bouillon, and claims that this antigen is devoid of non-specific action and yields a high percentage of positive results. Wang and Crocket, who have more recently investigated the reaction, using a lipoid-free antigen, suggest that non-specific reactions may be due to the presence of lipoids in the antigen; they record 85 per cent. of positive reactions in clinically tuberculous cases.

**Whooping-cough.**—The deviation reaction with *B. pertussis* and the serum of infected cases was originally described by Bordet and Gengou as evidence of the pathogenicity of this organism. The results obtained by other observers have, however, shown a good deal of variation, and the reaction is certainly not an early feature of the disease. Its only practical application is, therefore, in the diagnosis of atypical cases.

**Malaria.**—A complement-deviation test has recently been described (J. Gordon Thomson) for the diagnosis of chronic cases of malaria in which parasites may be difficult to detect in the peripheral blood. The antigen is prepared from artificial cultures of the malaria parasites. Further observation is required to establish the value of the test in routine diagnosis.

In **relapsing fever**, after the second recrudescence, a specific complement-deviation reaction is obtained with the patient's serum and an extract of blood-clot from infected animals. This reaction has been advocated as a diagnostic test, and also for the differentiation of the various types of relapsing fever spirochaetes.

In **cerebro-spinal meningitis** the spinal fluid contains antigenic bodies which react with a meningococcus serum and produce a specific complement-absorption effect. This reaction has been used for diagnosis.

**Helminthiasis.**—Complement-deviating antibodies have been noted also in the blood of men and animals infected with various helminths. The antigen used for their detection is an extract of the parasite or of the cyst fluid in the case of *Tania echinococcus*. It is uncertain whether these reactions are sufficiently specific for diagnostic work. Promising results have been obtained in the diagnosis of schistosoma infection by using the patient's serum along with an antigen prepared by extracting with alcohol the parasite-containing livers of infested snails (*Fairley's reaction*).

**Identification and classification of bacteria.**—The complement-deviation reaction has been found of great value along with agglutination tests in the identification of various bacterial species by means of specific antisera and in the classification of allied organisms, e.g. coli-typhoid group, food-poisoning group, vibrios, diphtheroid organisms. If complement-absorption tests are used in this way, specific action must be clearly distinguished from group effects by quantitative methods. Absorption tests also aid in the clear differentiation of closely related organisms; saturation of a typhoid antiserum with *B. paratyphosus-B* removes the group antistances for this organism and leaves unaltered the specific antibodies.

**Identification of blood-stains.**—The complement-deviation reaction represents a more delicate test for the identification of the blood or serum of a particular animal than the precipitin test, which has already been described, and is frequently used in routine medico-legal examination of blood-stains. Exceedingly minute amounts of serum can be detected in this way, e.g. 0.00001 c.c. The same method can be applied in the detection of meat adulteration (*see* Precipitin Tests, p. 185).

### OPSONIC TESTS AND OPSONIC INDEX

The serum opsonins act on bacteria by rendering them susceptible to phagocytosis. Normal serum is active in virtue of the normal non-specific opsonin; an immune serum is characterized by a specific immune opsonin. The opsonic power of the serum for a particular

## SEROLOGICAL DIAGNOSIS

organism can be estimated and compared with that of normal serum as an indication of immunity-response in the course of an infection or as the result of artificial inoculation.

**Method.**—Blood specimens (obtained as for the Widal reaction, p. 182) are taken from the case to be tested and from one or more normal individuals. The serums are separated by centrifugalization. If several normal serums are available they are pooled; leucocytes are obtained by adding blood from a puncture of the finger to citrate solution, and centrifugalizing to deposit the corpuscles, which are then washed several times with salt solution; after the final washing and centrifugalization a "leucocyte cream" can be obtained by pipetting off the surface layer of the blood sediment, in which the leucocytes tend to accumulate. The organisms to be tested are prepared in the form of a saline suspension from a young culture and are killed by heat. Equal volumes of organisms, patient's serum, and leucocytes are drawn into a capillary tube, which is sealed in a flame and incubated for a quarter of an hour at 37° C. A similar mixture of the normal serum, leucocytes, and organisms is also incubated. After incubation, films are made from the mixtures and stained with Leishman's fluid. The number of organisms ingested by a certain number of leucocytes (e.g. 50) is counted in both specimens.

The opsonic index is the ratio: number of organisms ingested by 50 leucocytes with patient's serum, divided by the number of organisms ingested by 50 leucocytes with normal serum.

The "normal" index determined in this way is found to vary from 0.8 to 1.2, and any variation beyond this range is of pathological significance. A high index suggests a state of relative immunity, e.g. in the case of an infection or as the result of artificial immunization; a low index indicates that the resistance is reduced (negative phase); this may be due to increased absorption of opsonins by the organisms or their products, e.g. during an infection or as the result of a large dose of vaccine.

The test is always carried out with polymorphonuclear leucocytes, but it is doubtful if the effect of these cells also represents the action of the other phagocytic cells which may play an important part in resisting certain infections. The test nevertheless affords some comparison between the action of the pathological serum and normal serums.

In diagnosis, the demonstration of an abnormal index with one organism while the index with another is within normal limits is suggestive of infection with the former. Opsonic counts have also been utilized in controlling vaccine treatment, the dosage being regulated so as to avoid an excessive negative phase after the injection.

The reaction has also been employed with a view to prognosis; massage or exercise of an infected part is liable to liberate bacterial products from the lesion into the blood-stream with an immediate fall in the opsonic index; in this way it is possible to ascertain whether a local lesion—e.g. a tuberculous joint—has become inactive.

### THE ABDERHALDEN PREGNANCY REACTION

According to Abderhalden, when foreign substances—e.g. proteins or carbohydrates—are injected parenterally, specific antagonistic ferments are developed which can be demonstrated in the blood-serum and decompose the substance as in the case of enteral digestion. In pregnancy it was supposed that the foetal elements of the placenta, being foreign to the tissues of the mother, led to the development of sero-enzymes which could digest the placental protein *in vitro*.

The detection of these ferments can be carried out by demonstrating the formation of amino-acids as the result of interaction of the serum and boiled placenta; the placental material and serum are placed in a dialysing shell and the diffusible amino-acids can be tested for in the external fluid by the violet colour obtained with ninhydrin (triketohydrinden hydrate).

Though an emphatic positive reaction is frequently obtained with the serum of pregnant women, the reaction can hardly be regarded as a specific test for pregnancy.

### ALLERGIC REACTIONS

These reactions depend on the hypersensitiveness of the tissues to the products of the infecting organism, and have been demonstrated in various conditions—e.g. tuberculosis, syphilis, typhoid fever, ringworm, glanders—particularly in chronic infections. In early cases the tests are more likely to yield negative results.

**Tuberculin reactions.**—Koch's "old tuberculin" is generally used for these reactions. It represents the products of the tubercle bacillus, and is prepared from glycerin-bouillon cultures heated at 100° C. for one hour, fil-

## SEROLOGICAL DIAGNOSIS

tered, and evaporated to one-tenth of the original volume. The preparation consists of the soluble products of the organism together with certain constituents of the medium, glycerin, peptone, etc., and some of the effects of tuberculin have been ascribed to these constituents. The reaction may be carried out by (1) subcutaneous injection, (2) dropping a dilute solution into the conjunctiva (Calmette), (3) applying tuberculin to an abraded skin area (von Pirquet), (4) intracutaneous (or intradermal) injection, (5) applying tuberculin to the skin in ointment.

**Subcutaneous method.**—Old tuberculin ( $\frac{1}{2}$  mg. diluted with 1 c.c. normal saline) is injected subcutaneously. In the tuberculous subject the administration is followed after twenty hours by a general reaction—pyrexia  $102^{\circ}$ – $103^{\circ}$  F. (a rise of temperature over  $1^{\circ}$  F. is significant), and also a local reaction—swelling, pain, and tenderness at the site of injection. At the same time a “focal” reaction may occur in the existing lesion, e.g. lupus lesions after a few hours show acute inflammatory changes. If the lesion is a pulmonary one there may be temporary aggravation of the condition as shown by physical signs; in the case of other internal lesions also—e.g. of the kidney—pain may be experienced locally, and these focal effects may aid in diagnosis by locating the lesion.

If no reaction occurs, the test should be repeated two or three times at intervals of four days, as a subsequent test may elicit a positive result, and the dose may be increased. If a slight pyrexial reaction is noted—e.g.  $0.5^{\circ}$  F. rise with also a local effect—the subsequent dose should be the same as the first, and will frequently elicit a more conclusive result. In the case of infants smaller doses are given, e.g.  $\frac{1}{2}$  mg.

Non-specific reactions, especially following large doses, have been frequently noted, i.e. in apparently non-tuberculous cases, but the question arises whether these reactions may not be due to latent tuberculosis, or to an old infection.

If minute doses are followed by definite reactions the result is significant of the existence of an active lesion.

The subcutaneous test should not be applied in febrile cases, general tuberculosis, advanced lesions, possible tuberculosis of the larynx, ear, or meninges, pregnancy, or cases with symptoms of nephritis, diabetes, or heart disease. It must be borne in mind also that the focal

reaction may be prejudicial, e.g. in early tubercle of the lung, and may produce extension of the lesion; on this account the subcutaneous test is not frequently used.

**Calmette's ophthalmic reaction.**—A few drops of a 1-per-cent. dilution of tuberculin in saline are instilled into the conjunctiva. A positive result consists in a marked inflammatory reaction of the conjunctiva after about twenty-four hours. The effect may be severe in some cases, and may lead to damage of the cornea. It is doubtful if this test should ever be applied as a routine procedure, in spite of the valuable diagnostic results it has elicited.

**Von Pirquet's cutaneous reaction.**—The skin of the forearm is cleansed with spirit, and then three abrasions are made about 2 in. apart with a special “borer.” On two of these a drop of tuberculin is placed and allowed to act for a quarter of an hour: one is left uninoculated as a control. In a positively reacting case the tuberculin-inoculated abrasions, after forty-eight hours, develop papular infiltrated swelling with a surrounding area of hyperæmia. Negative results are of significance in excluding a tuberculous infection, but it is doubtful if positive reactions are to be regarded as indicative of active tuberculosis. Thus, apparently non-specific reactions are frequently noted, and these may be due to a latent infection of no clinical importance.

**Intracutaneous test.**—0.005 mg. of old tuberculin in 0.05 c.c. of saline solution is injected into the skin of the forearm by means of a syringe with a fine needle. The reaction consists of an inflammatory area with infiltration at the site of injection, reaching its maximum after twenty-four to forty-eight hours.

**Moro's test.**—Old tuberculin incorporated in anhydrous lanolin (5 c.c. of tuberculin to 5 gm. of lanolin) is applied to the skin (of the abdomen) by inunction. A positive result consists in the eruption of papules after twenty-four hours.

A tuberculin prepared from the bovine type of tubercle bacillus has also been used to differentiate infections of this type. Insusceptibility to tuberculin may develop after a course of tuberculin treatment. The intracutaneous and subcutaneous methods may be regarded as the most reliable of these tuberculin tests.

**“Luetin” reaction (Noguchi).**—Luetin is prepared from mixed cultures of a number of strains of the *Spirillum pallidum* killed by

## SEROLOGICAL DIAGNOSIS

heat. The reaction is carried out by injecting intracutaneously 0.035 c.c. of the preparation with an equal volume of sterile salt solution. In negative cases, after twenty-four hours there may be a slight inflammatory reaction, which quickly disappears. The positive reaction usually consists of a well-marked infiltrated papule which appears in twenty-four to forty-eight hours and attains its maximum in four or five days. In some cases the papule may become vesicular and even pustular. Occasionally a "torpid" reaction is noted, i.e. the development of a pustule after a latent period of as long as a fortnight following the injection. Positive reactions are not characteristic of the primary and secondary stages of syphilis, but a large proportion of tertiary and latent cases react to luetin, and in such cases the luetin test may be positive though the Wassermann reaction is negative. Thus, the luetin reaction is of great value in the diagnosis of those cases of syphilis which fail to exhibit a positive Wassermann reaction.

**Allergic reaction in typhoid fever.**—A test analogous to Calmette's tuberculin reaction has been used; "typho-protein" prepared from a number of strains of *B. typhosus* is instilled into the conjunctiva. The test is regarded as specific (Austrian), and may be positive before the Widal reaction has developed. Cutaneous and intracutaneous reactions with "typhoidin" (prepared from cultures of *B. typhosus*) have been regarded as indicating immunity against typhoid infection, and positive results are obtained in a large proportion of convalescent typhoids and in typhoid-inoculated persons.

Allergic reactions have also been observed in gonorrhoeal infections.

Cutaneous and intracutaneous allergic reactions have been employed for demonstrating hypersensitiveness to various proteins, e.g. horse-serum, milk, etc. (See Anaphylaxis, under IMMUNITY.)

### SCHICK'S TEST FOR NATURAL IMMUNITY TO DIPHTHERIA

Certain normal persons may possess sufficient natural diphtheria antitoxin to protect them from a diphtheria infection. This can be gauged by means of Schick's test:  $\frac{1}{80}$  of a minimum hæmolytic dose (see IMMUNITY) of diphtheria toxin is injected intracutaneously; the dose of toxin is made up in 0.1 c.c. of salt solution. After twenty-four hours, in the non-immune person an area of hyperæmia and

## SEXUAL FUNCTIONS, MALE

induration develops at the site of injection, reaching its maximum after about forty-eight hours. A positive reaction indicates the need for immunization in persons exposed to infection.

T. J. MACKIE.

**SEROUS MENINGITIS (SEROUS APOPLEXY)** (see MENINGITIS).

**SERUM DISEASE (ANAPHYLAXIS)** (see IMMUNITY).

**SERUM-THERAPY** (see IMMUNITY).

**SEVEN-DAY FEVER** (see RELAPSING FEVER).

**SEXUAL FUNCTIONS, MALE, DISTURBANCES OF.**—The chief disturbances of the male sexual functions—apart from sterility, which is dealt with in a separate article (see STERILITY, MALE)—are impotence, spermatorrhœa, nocturnal emissions, and priapism.

**Impotence.**—This may be a result of many different conditions, and its cause in each case must be carefully distinguished.

1. It may be due to *malformations and local injuries and diseases of the genital organs*, as a rudimentary penis, hypospadias or epispadias, congenital curvature of the penis with shortness of the frænum, tight prepuce, lack of development or acquired atrophy of the testes due to any cause, epididymitis and orchitis from mumps and other conditions, chronic prostatitis and prostaticorrhœa; and occasionally it is associated with a large scrotal hernia or hydrocele. After prostatectomy, desire or the power of erection may be lost, or there may be no emission of seminal fluid.

The **treatment** of impotence due to any of these causes should be directed to the original disease.

2. *Diseases of the ductless glands* frequently induce impotence, and particularly diminished function of the thyroid and pituitary. A male with myxœdema usually loses desire and the power of erection, and in states of hyperthyroidism there may be partial impotence owing to incomplete erection or premature emission. In myxœdematous women desire and voluptuous sensation disappear. Pituitary diseases, especially those associated with symptoms of dystrophia adiposo-genitalis, also entail impotence. The sexual instinct is largely dependent on the interstitial cells of the testes, and if these atrophy impotence results.

## SEXUAL FUNCTIONS, MALE, DISTURBANCES OF

3. *Impotence as a symptom of other diseases* is not uncommon. It often occurs in diabetes mellitus and Bright's disease, and sometimes in phthisis, though in the latter sexual desire is often increased. It occasionally follows cranial and spinal concussions, and is common in many nervous diseases, as tabes and spastic paraplegia; in paraplegia erections may be too frequent, but the power of penetration and ejaculation may fail. It is often associated with melancholia and other mental disorders. Addiction to drugs, as opium, morphia, alcohol, and tobacco, can diminish or abolish sexual power; it is well known that alcohol "provokes the desire but takes away the performance," and chronic alcoholics are often impotent. Excessive smoking may produce the same effect for a time in young men.

The **treatment** of impotence due to these conditions is difficult and usually ineffective, but the trouble is, as a rule, of little consequence, since the desire usually disappears too, and the condition therefore rarely disturbs the patient. In the case of drug-takers, potency may be regained by cutting off the drugs and improving the health by tonic treatment.

4. *Nervous or psychological impotence* is a more distressing symptom, as the desire generally persists and the loss of power is frequently so distressing that the patient becomes a sexual hypochondriac. It is most commonly a symptom of neurasthenia brought on by mental overwork or worry, anxieties, emotional disturbances, or ill-health associated with disturbing preoccupations. It is often seen in neurotic patients and persons of neuropathic stock who indulged in sexual excesses or in masturbation in earlier life, or who had acquired some form of venereal disease. It is not, however, directly due to these; its usual mode of origin is that the neuropathic patient who is about to marry, or whose attention is otherwise directed to his sexual functions, begins to worry and to suffer remorse for his moral lapses, then the idea of impotence arises and becomes an obsession that is rarely absent from the mind, so that he eventually becomes a sexual hypochondriac. And as no other bodily function is so dependent on ideas and mental attitudes as the sexual, a real impotence may develop. This often takes the form of premature emissions—that is, emission before penetration is achieved or before full satisfaction is obtained. This symptom is not uncommon in newly married men, especially in those of poor physical stamina or feeble sexual

power. Profound exhaustion after coitus is also often complained of by neurasthenic and debilitated persons.

The **treatment** of nervous impotence should in the first place be directed to improving the general health and physical condition by open-air exercises and games, cold baths and douches, and a generous diet. Stimulants should be withheld, and it is advisable to cut off tobacco. Tonics, especially phosphorus and the hypophosphites, are usually recommended. The commonly prescribed aphrodisiacs are of little use, but Lane recommends the following pill:

Ry Extr. damianæ gr. ii.  
 Lecithini gr. i.  
 Extr. nuc. vom. gr. ½.  
 Yohimbina hydrochl. gr. ½.  
 T.d.s

Testicular extracts, too, as spermin, are sometimes useful. The value of suggestion in cases of nervous impotence cannot be too strongly emphasized; a candid explanatory conversation is often sufficient, but stronger forms of suggestion, or hypnotism, may be necessary.

**Priapism.**—A persisting erection that is often painful may be the result of spinal disease, especially of those forms that produce paraplegia, or of prostatic enlargement. It is also found in leukaemia and gout, and occasionally it occurs without any apparent cause.

**Treatment.**—Cold applications with large doses of bromide or camphor usually give relief. If the condition is due to reflex irritation the application of cocaine to the glans penis may be of service. In more troublesome cases an epidural injection by Cathelin's method, or a lumbar puncture and the intrathecal injection of a small quantity of stovaine, may be required.

**Spermatorrhœa.**—The involuntary escape of semen, unattended by erection, desire, or voluptuous sensations, is most commonly a symptom of sexual neurasthenia, but it may be due to overdistension of the seminal vesicles, to chronic gonorrhœa, or to irritability of the nervous apparatus. The escape generally occurs with micturition or defæcation, or it may be due to local irritations, as in riding or cycling, or to emotional disturbances or anxiety states.

**Treatment.**—Tonics and attention to the general health and physical condition are required, and any local condition that may have an influence should be remedied.

**Nocturnal emissions.**—These occur normally at variable intervals in healthy young men,



## SHOCK AND COLLAPSE

but when too frequent or too excessive have a debilitating effect. The emissions generally take place in the early morning, when the bladder is full, and constipation has often some influence on them. The erotic dreams that accompany them may be their cause, but are usually a result of the physiological emission of the semen.

**Treatment.**—Emissions require treatment only when excessive; a dose of bromide at night, and a healthy outdoor life with plenty of physical exercise during the day, are generally sufficient. Care should also be taken to avoid distension of the bladder and constipation.

GORDON HOLMES.

**SHINGLES** (see HERPES ZOSTER).

**SHOCK AND COLLAPSE.**—The terms "shock" and "collapse" have been used somewhat vaguely to describe conditions of depressed vitality of fairly rapid onset. The difficulty of definition has been accentuated by the fact that they have been used in alternative senses by different writers; what some have called "shock" others have called "collapse," and vice versa. It seems clear that by "shock" the earlier writers usually meant a condition of sudden onset, resulting from injury, pain, fright, or violent emotion, and characterized by more or less complete loss of consciousness, with a feeble circulation and respiration. In other words, "shock," as so understood, differed from "fainting" only in severity and persistence. There is no difference of opinion as to the nervous origin of this type of effect. It is essentially a reflex inhibition, the heart being inhibited through the vagus nerves, the tone of the arteries by reflex action through the vaso-motor centre, and the effect on the respiratory centre being probably, for the most part, a secondary result of the failing circulation. In such a condition the blood tends by gravity to accumulate in the relaxed vessels of the splanchnic area, so that mere posture has a large influence on the genesis or the relief of the condition. "Collapse," on the other hand, was the term applied to a more persistent and progressive defect of the circulation, which might arise from a variety of causes: it might follow a prolonged and unrelieved "shock" or might be the result of very severe hæmorrhage, of poisoning, or of infection.

In recent years, largely under the influence of Crile, there has been a tendency to invert

the application of these terms. The sudden inhibitory phenomenon Crile called "collapse," reserving the term "shock" for the condition which he could produce in anæsthetized animals by various kinds of traumatism, and which in its general features undoubtedly corresponded more closely with what had earlier been called collapse. This use of the nomenclature has persisted in most subsequent writings based on experimental work, and the whole controversy which has arisen concerning the nature of "shock" has centred round the physiological nature of this persistent, progressive circulatory deficiency.

During the late War surgeons studying the effects following injuries again distinguished two conditions. One was of rapid onset, obviously of nervous origin, and was characterized by cardiac and vaso-motor inhibition. It might be, but seldom was, directly fatal. Usually the wounded man recovered from this condition. It was suggested, with good show of reason, that it represented a protective reflex, the low blood-pressure tending to stop further hæmorrhage until the wounded vessels became closed by thrombi. At a later stage another and more serious condition might develop, which superficially resembled the first, both being due to failure of effective circulation, but which showed no tendency to spontaneous betterment, becoming progressively more severe and ending fatally in default of efficient treatment. Military surgeons, in writing of these conditions, have avoided the ambiguity of the terms "shock" and "collapse," and have distinguished them as "primary shock" and "secondary shock."

Before considering the treatment of these conditions it is necessary to consider the nature and cause of secondary shock in greater detail. It is agreed that the central feature of the condition is the deficiency of the circulation, and that this deficiency is not due to direct weakening or inhibition of the heart's activity, but to defect of output from the left side, owing to inadequate return of the blood to the right side of the heart. So far as the output of the heart is concerned, the effect is very similar to that caused by a severe hæmorrhage. But concerning the cause of this failure of the return of blood to the right auricle, opinion is still divided. There are some who, following Crile, ascribe it to fatigue of the bulbar centres as a consequence of overstimulation. The result of this, it is suggested, is to produce slowly and persistently an effect

## SHOCK AND COLLAPSE

on the distribution of the blood similar to that which, in primary shock, is produced by inhibition in a more rapid and evanescent form: the arteries in the splanchnic area gradually lose their tone as the vaso-motor centres become fatigued, the blood tending to be pooled in that region instead of returning normally to the heart. According to this view the low blood-pressure causes absorption into the vessels of fluid from the tissues, so that the blood is more dilute and the volume in the system actually increased, though the volume reaching the heart and expelled by it at each beat is so seriously diminished. The alternative view, for which evidence accumulated during the War, regards a diminution of the volume of blood in effective circulation—an "oligæmia"—as the central feature of the condition. This is accounted for partly by a loss of plasma from the vessels, producing a concentration in respect of corpuscles, diminution of volume, and increase of viscosity; partly by a loss of blood from currency owing to its stagnation at the periphery in relaxed capillaries.

Several theories again have been advanced as to the cause of this condition of partially stagnant circulation, each giving rise to suggestions for appropriate treatment. Some have held that it consists in a deficiency of carbon dioxide in the blood, resulting from over-activity of the respiratory centre in response to sensory stimuli. The administration of air or oxygen mixed with an excess of carbon dioxide has accordingly been recommended. Others have regarded the depletion of the alkali-reserve of the plasma, which undoubtedly occurs in this as in any condition of defective circulation, as the cause of the progressive failure. They have accordingly recommended the injection of solutions of sodium bicarbonate into the blood-stream. The all-too-abundant opportunities which the War afforded for studying cases of "secondary shock" produced no convincing evidence in favour of either of these views. Yet others have regarded emboli of fat, liberated from damaged tissues into the blood-stream, and obstructing pulmonary capillaries, as a most important cause of the condition. There is no room for doubt that this form of embolism can be produced by violence, and the symptoms which it sometimes causes are such as might easily be confused with those of secondary shock. Experience during the War, however, failed to justify the attribution to fat-embolism of a general

significance as the cause of this type of condition.

The tendency of much recent observation has been to indicate the importance of a factor of a different kind, namely, the absorption from injured tissues of the toxic products of autolytic change. There is no doubt that cleavage products produced from proteins by the action of ferments or bacteria can produce a condition in many ways resembling the secondary shock described by military surgeons. The effect appears to be due to a weakening of the normal tone of the capillaries and to an abnormal permeability of their walls, so that the blood tends to stagnate at the periphery and its plasma to leave the system. The recognition of this factor brings secondary shock into relation with the state of collapse produced by severe bacterial toxæmia, such as occurs in cholera.

While there is a good deal of evidence in favour of regarding such a traumatic toxæmia as the central factor of secondary shock following wounds in war, many contributory factors have been recognized. Hemorrhage, a complication present in the vast majority of cases, would obviously accentuate a condition due to lack of blood in effective currency. Cold, which of itself has a depressant effect on capillary tone; lack of fluids, impairing the natural power of restoring the blood-volume by absorption from the tissues; anæsthetics such as chloroform and ether, which have been shown experimentally to accentuate the effect of toxic products from the tissues; fatigue and pain, which by exhausting the vaso-motor centre weaken its effort to compensate for deficient blood-volume by arterial constriction—all these probably play the part of accessories, and have an important bearing on preventive and remedial treatment.

The problem of most immediate interest is to discover how far the conclusions derived from the study of shock in war are applicable to the conditions arising in civilian life, to which that term has been applied. As regards the "primary" shock, no special difficulty arises. The condition of inhibition following the accidents of peace differs in no important respect from that following wounds in war. The treatment is on obvious and well-recognized lines—the recumbent position, with the foot of the couch or stretcher raised, to neutralize the tendency of the blood to accumulate in relaxed abdominal vessels; removal of any impediment to or restriction of respiration;

## SHOCK AND COLLAPSE

the application of external heat; massage of the limbs to assist the return of blood to the heart. Effort should, of course, be made to secure torn vessels of any size before restoration of the blood-pressure, which might otherwise be accompanied by renewed hæmorrhage. Extract of the infundibular lobe of the pituitary body, injected into the muscles or into a vein, is often of the greatest value.

There are doubtless cases also in civil life, resulting from severe injury, in which, on account of much hæmorrhage or failure of the restorative reaction, a primary shock tends to merge into a secondary shock similar to that seen in war. The position is more doubtful with regard to the important condition known as "surgical shock" or "postoperative shock." There are those who, while admitting the toxæmic element in the wound-shock of war, would maintain that the shock following operation is essentially a fatigue of the nerve-centres. Fortunately, the conception adopted as to the precise mechanism by which the condition is produced does not seriously affect the indications for treatment.

It is now generally admitted that the proper **treatment of surgical shock** is its prevention. Recent experience has given some indications of value in this direction. It is clearly desirable that the system should not be depleted of fluid in anticipation of operation, and the fasting and purgation, which are items of the usual preparatory routine, have undoubtedly often been carried to excess. Maintenance of the body temperature during operation is an important point, to which insufficient attention is frequently given. The amount of blood lost by hæmorrhage during operation is habitually under-estimated, and some recent actual measurements have given surprising results. The effect of anæsthetics such as ether and chloroform needs more careful consideration than it has hitherto received. Experience during the War has emphasized the serious danger attending their use in cases which are already toxæmic. Crile, on other grounds, had advocated the substitution of nerve-blocking with local anæsthetics (anoci-association), and nitrous oxide to produce unconsciousness, as much less liable to produce shock. It is not improbable that the absence of ether or chloroform was a more significant factor in the success of his method than the complete cutting-off of the impulses from the field of operation to the nerve-centres, to which he attributed importance. In any case,

## SIGMOIDOSCOPY

it cannot be doubted that, when shock is to be feared, nitrous oxide with oxygen is the anæsthetic of choice, combined, if necessary, with local anæsthesia to secure immobility.

If, in spite of precaution, surgical shock appears, its treatment must depend to some extent on the precise nature of the condition. If arterial relaxation, whether due to inhibition or to fatigue of the nerve-centres, is an important factor, the pituitary (infundibular) extract is likely to do good, and might in any case be given a trial. Strychnine and alcohol, formerly the recognized remedies, are fairly generally admitted to possess no value. If the condition is chiefly a toxæmic oligæmia, pituitary extract is unlikely to have any value. The treatment must be directed to restoring the volume of the circulation. Warmth, and water in large quantities—by the mouth if the patient can retain it, otherwise by the rectum—should be given a good trial, but not for too long. The condition, if not soon relieved, is liable to pass beyond remedy, and, if the patient does not soon show signs of rallying, recourse should be had to intravenous injection. For this purpose saline solutions, such as Ringer's fluid, with or without adrenalin, which previously were in general use, became thoroughly discredited during the War. The effect is only temporary and often the patient is then worse than before, the additional fluid rapidly leaving the system either by the kidneys or into the tissues. The case should be treated like one of hæmorrhage, and, whether blood has actually been lost from the body or not, human blood should be infused if obtainable (*see* TRANSFUSION), or, failing that, a saline solution containing an osmotically active colloid such as gum acacia. The solution recommended by Bayliss contains 6 per cent. of the gum with 0.9 per cent. of sodium chloride. If a compatible donor can be obtained, transfusion of unaltered blood can be carried out with Kimpton's paraffined collecting-tube, or the blood can be mixed with solution of sodium citrate and glucose. H. H. DALE.

**SIALORRHOEA** (*see* SALIVA, ANOMALIES OF SECRETION OF).

**SIDEROSIS** (*see* PNEUMONOCOINOSSES).

**SIGMOIDOSCOPY.**—The whole rectal canal and the lower portion of the sigmoid loop can be examined by a sigmoidoscope. This instrument is a straight, hollow, plated metal tube, just over a foot long. It is provided

## SIGMOIDOSCOPY

with an obturator which enables it to be passed through the anus without pain or difficulty. The illumination is provided by a small electric light which is passed to the inner end of the tube and is attached to a thin rod fixed to a fitting at the observation end of the tube. The electrodes are attached to the fitting; this also has a side opening (controlled by a tap) to which the rubber tube of an inflation pump can be affixed. A glass eyepiece is fitted at the end. Long narrow sponge-holders are provided. (Fig. 89.)

**Position of the patient.**—Either the lateral, the lateral-prone, or the lithotomy position may be used. When little assistance is available the lateral-prone position is probably the best.

**Anæsthetic.**—The examination may be conducted without anæsthesia, but in sensitive subjects it is better to give a general anæsthetic.

**Technique.**—The tube is fitted with the obturator; the rounded end is well lubricated with vaselin or oil, and gently introduced through the anal opening. So soon as the end is well in the canal the obturator is withdrawn, the light introduced, the electrodes are attached to the fitting, the rubber tube of the inflator is affixed, and the eyepiece put on.

Before pushing the tube any farther up the rectum the canal is inflated gently by squeezing the inflator. The electric light is turned on, and the observer looks through the eyepiece and pushes the tube on into the lumen of the canal as it opens out under inflation. In no circumstances must the tube be pushed on without seeing clearly the lumen into which it is to pass.

The tube can be passed up 10–12 in. If any mucus or faecal material obscures the view, it must be wiped away by a piece of gauze or wool introduced by the sponge-holder.

**Indications for use of the sigmoidoscope.**—Affections of the anal canal are well enough observed with one of the various forms of small proctoscope or speculum. In any doubtful

case with rectal symptoms, i.e. the passage of blood or mucus, rectal discharge, chronic diarrhoea, increasing and obstinate constipation, alternate attacks of diarrhoea and constipation, sigmoidoscopy will be of use. By this means a small piece of any rectal growth can be removed for microscopy, or a topical application may be made to an ulcer on the rectal wall. Strictures in the lower sigmoid and ulceration high up in the rectum can be observed.

**Appearance of the normal rectum.**—The normal rectal mucosa is dark red in appearance, and the lumen of the canal easily dis-

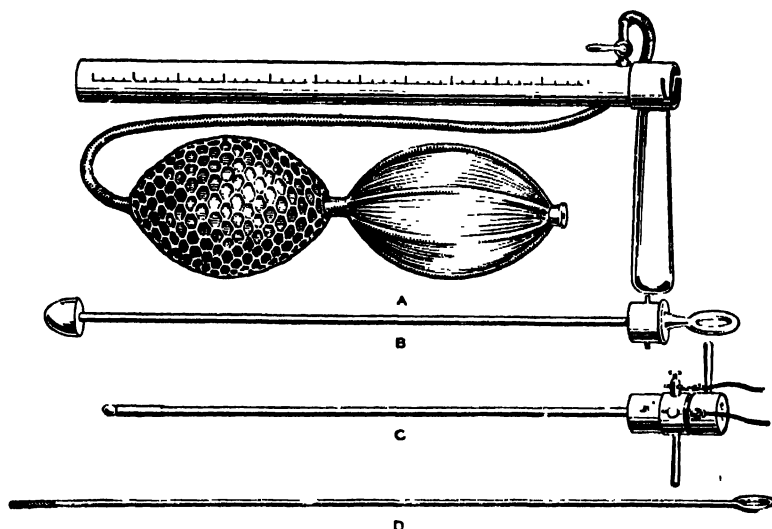


Fig. 89.—The sigmoidoscope.

A, Metal graduated tube with hand-bellows attached; B, the obturator; C, rod carrying the light at distal, and the terminals at proximal end; D, the applicator.

tends as the inflator is used. The folds of Houston can be readily identified. When the lumen does not dilate, either the end of the tube is pressed too closely against the rectal wall or a stricture is present.

The important points to remember in the use of the sigmoidoscope are that one should never push the instrument on without seeing the passage clear, and that force should never be used.

ZACHARY COPE.

**SILICOOSIS** (see PNEUMONOCOINOSSES).

**SINGER'S NODES** (see Chronic Laryngitis, under LARYNGITIS).

**SINUS ARRHYTHMIA** (see HEART-BEAT, ABNORMALITIES OF).

## SINUSES

**SINUSES.**—Narrow tubular tracts opening on the surface of the body at one end, and leading down to a focus of infection at the other. Sinuses fail to heal for several reasons. There may be stagnation of discharge so that infected exudations become pent up and prevent repair. There may be constant reinfection, or too much movement to allow of closure, as in sinuses in the ano-rectal region. There may be a foreign body, an unabsorbed ligature, or a dead piece of bone at the bottom of the sinus, or there may exist a tuberculous infection of its walls. Tuberculous sinuses are very difficult to heal. They may frequently be distinguished clinically because their orifices are surrounded by bluish, often undermined, skin and the discharge is watery, whereas in a septic sinus the mouth is usually surrounded by red pouting granulations, and thick pus exudes. The downgrowth of epithelium from the mouth of a sinus will effectually prevent closure.

**Treatment.** Rest is important, and is obtained, according to the situation of the sinus, by splinting a limb, the application of a thick layer of wool and bandage to the neck, or the division of the anal sphincter. Next, it is important to search for a foreign body or necrosed piece of bone at the bottom of the sinus; this must be done under anaesthesia. At the same time free drainage must, if possible, be provided by converting the tunnel into an open groove. A postoperative sinus due to the retention of an infected suture will always close when this is removed. If epithelium is spreading from the surface over the walls of the sinus it must be curetted away. After any operation the wound should be packed with gauze to induce healing from the bottom.

*Beck's bismuth paste injection* is useful both for tracing the course of a fistula by skiagram and as a curative measure. When injecting a sinus, place the patient so that the mouth of the sinus lies uppermost. This will allow the air in it to escape. After the sinus has been injected, a dry sterile dressing should be applied. A good deal of the paste will have escaped in twenty-four hours. It is not intended to be retained, and if it has not come away from a large sinus, such as that associated with an unhealed empyema cavity, in five days, it should be drawn out by suction. Several injections may be necessary to attain success, but when the purulent discharge has been replaced by a serous exudation it is not

## SINUSES, ACCESSORY AIR

necessary to reinject, for the sinus will usually heal. Beck's method is useful in tuberculous as well as septic sinuses, but it is useless to employ it if there is a foreign body in the sinus. Apparently it acts by setting up an intense leucocytosis in the walls of the sinus, by which the infecting organisms are killed.

Ionization with zinc ions has been used for sinuses with a certain amount of success. The anode, made of a zinc rod, is thrust into the sinus, filled with 1-per-cent. zinc sulphate or packed with gauze soaked in this solution. A weak current of 2-10 ma. is sufficient; it should be passed for fifteen to twenty minutes daily.

C. A. PANNETT.

**SINUSES, ACCESSORY AIR, DISEASES OF.**—Under this head will be considered—

1. Diseases of the Frontal Sinus.
2. Diseases of the Ethmoidal Cells.
3. Diseases of the Maxillary Antrum.
4. Diseases of the Sphenoidal Sinus.

### 1. DISEASES OF THE FRONTAL SINUS

**ACUTE SUPPURATION.**—Any acute or chronic inflammatory condition in the nose may cause acute suppuration of the sinus by direct extension into it; this is especially apt to occur in the acute rhinitis associated with such diseases as influenza and scarlet fever. Some cases follow direct injury to the sinus.

**Symptoms.**—Pain and a feeling of tension in the sinus always occur, and frequently also severe frontal headache; pressure on the anterior wall of the sinus or on the inner angle of the orbit will usually elicit intense pain. A purulent discharge from the affected nostril is complained of by some patients; sometimes this discharge is intermittent in character, and with each appearance of pus the local symptoms may be slightly relieved.

In some cases redness and oedema appear over the affected sinus and in the upper eyelid on the same side; and occasionally even an abscess is found over the anterior wall of the sinus.

The temperature is always raised, the fever being accompanied by general malaise and perhaps by rigors.

**Diagnosis.**—Examination shows that the nasal mucous membrane on the side affected is in a state of acute inflammation, which is especially severe over the anterior end of the middle turbinal bone. If the opening of the sinus is not obstructed, pus is seen passing

## SINUSES, ACCESSORY AIR, DISEASES OF

from under the anterior end of the middle down over the body of the inferior turbinal bone.

**Treatment.**—If the disease is encountered in the early stage, rest in bed, combined with frequent inhalations of menthol, eucalyptus, and compound tincture of benzoin, often gives relief. The anterior end of the middle turbinal bone should be removed at once under cocaine or a general anæsthetic, as its removal not only assists in the free escape of discharge but also enables the frontal-sinus catheter to be inserted more easily. The sinus should be washed out once daily, or more often if necessary, until the malady is cured.

When the symptoms show no sign of abatement, when they are increasing, or when there is definite swelling or œdema over the sinus, it should be opened up at once. The operation is a dangerous one, and, if it is not carried out skilfully, osteo-myelitis of the frontal bone or intracranial complications are very apt to occur; the general practitioner without special experience is therefore advised not to attempt this operation.

If seen in its early stages and treated energetically the condition usually settles down quickly. Some cases, however, develop septicæmia, thrombosis of the cavernous sinus secondary to complicating orbital cellulitis, or intracranial conditions which prove fatal in spite of operative treatment.

**CHRONIC SUPPURATION.**—The same conditions which produce acute suppuration in the sinus may lead to chronic suppuration.

**Symptoms.**—Nasal obstruction is complained of in most cases, owing to the enlargement of the anterior end of the middle and of the body of the inferior turbinal bones which usually takes place. A purulent discharge is also present; it is often greatest when the patient gets up, the pus which has collected in the sinus during the night being then able to escape. Frontal headache is usual and is most severe when there is obstruction to the free escape of discharge. Tenderness on firm pressure over the anterior wall of the sinus and over the inner part of the roof of the orbit may be present.

Most patients also complain of symptoms such as general malaise, pallor, pains in the joints, etc., due to the absorption of toxins.

As the ethmoidal cells and the antrum almost always become infected in the later stages of the disease, symptoms resulting from their involvement may be superadded.

**Diagnosis.**—It must be remembered that the presence of pus under the anterior end of the middle turbinal bone is diagnostic of suppuration in one or all of the anterior group of sinuses; to make an accurate diagnosis it is therefore necessary to wipe away any pus which may be present in this region and, after the application of cocaine, to insert a frontal-sinus catheter. Air should then be blown gently into the sinus, and if pus is present it will be seen to bubble out round the catheter.

If the anterior end of the middle turbinal bone is much enlarged and interferes with the passage of the catheter it should be removed.

Where the facilities exist, should there be any doubt in regard to the diagnosis, an X-ray photograph may be taken and is sometimes helpful, the diseased sinus being found to have a blurred outline when compared with the unaffected one.

**Treatment.**—The anterior end of the middle turbinal bone should always be removed and the sinus washed out daily with a weak antiseptic lotion; a large number of cases react well to this form of treatment.

When the symptoms persist or are obviously getting worse, surgical treatment must be undertaken.

A general practitioner who has not had considerable experience in operating for the radical cure of this condition is strongly advised not to attempt any of the various operations which have been devised, for the results might prove far from satisfactory.

**MUCOCELE.** This condition is occasionally met with, and usually appears as a painless soft swelling at the inner angle of one or both orbits. Fluctuation and even "egg-shell" crackling can sometimes be elicited. The eyeball is displaced downwards and outwards, and where the mucocele is large much deformity is produced. Complete excision is the only treatment.

### 2. DISEASES OF THE ETHMOIDAL CELLS

**ACUTE SUPPURATION.**—This is always caused by direct extension of some infective process from the nose.

**Symptoms.**—The symptoms most commonly complained of are pain and a feeling of tension at the inner angle of the orbit, accompanied by frontal or occipital headache. Nasal obstruction sometimes results from swelling of the inflamed nasal mucous mem-

## SINUSES, ACCESSORY AIR, DISEASES OF

brane, and a muco-purulent or purulent discharge is usually present. As a rule there is a rise of temperature accompanied by malaise. A frequent complication is orbital cellulitis, which manifests itself by the onset of œdema of the eyelids, displacement of the eyeball, chemosis and proptosis.

**Diagnosis.**—If the anterior group of cells is involved, pus is seen under the anterior end of the middle turbinal bone, whereas if the posterior group is affected it is seen passing down over the middle turbinal bone by anterior rhinoscopy, and over the posterior end of the same bone by an examination of the post-nasal space. The nasal mucous membrane is in all cases found to be in a state of acute inflammation.

**Treatment.**—Inhalations of compound tincture of benzoin and menthol should be ordered in all cases, and as much as possible of the middle turbinal bone removed to allow of as free drainage as possible.

When symptoms of orbital cellulitis are present it is necessary to open up the orbit at its inner angle and to deal with the ethmoidal cells by the combined orbital and nasal routes.

Most cases settle down quickly with appropriate treatment; some, however, become chronic, and, should the orbit become involved, blindness may follow, or thrombosis of the cavernous sinus or meningitis may cause the disease to terminate fatally.

**CHRONIC SUPPURATION.** Owing to the complicated nature of the ethmoidal cells, acute suppuration in this region is very apt to become subacute and finally chronic in character.

**Symptoms.**—Patients complain of a muco-purulent or purulent nasal or postnasal discharge accompanied by crust-formation; if polypi are present, or if there is enlargement of the turbinal bones, nasal obstruction occurs in varying degrees.

There may be frontal or occipital headaches, especially when polypi or enlargements of the middle turbinal bone prevent the free discharge of pus from the affected cells. Tenderness is present on pressure, in some cases, at the inner angle of the orbit.

Aural, pharyngeal, or laryngeal complications supervene sooner or later in the course of the disease. Both the antrum and the frontal sinus are very apt to become infected in the later stages.

**Diagnosis.**—Pus is seen in the positions described under Acute Suppuration. Either a

single polyp or polypi in large numbers may be present. The anterior ends of both the middle and inferior turbinal bones are usually enlarged, and crusts may be seen adherent to them.

When the disease has been present for a considerable time the turbinal bones become atrophied, and the conditions found by anterior and posterior rhinoscopy resemble those described under Atrophic Rhinitis in the article on RHINITIS.

**Treatment.**—All cases should be treated by the removal of as much as possible of the middle turbinal bone, and the breaking down of as many ethmoidal cells as are accessible, so as to establish free drainage. Following this, a douche of normal saline solution should be used morning and evening, followed by a spray of 5 per cent. menthol in paroline, or of the following:—

R̄ Menthol gr. viii.  
Ol. eucalyp. ℥i.iii.  
Ol. cinnam. ℥i.iii.  
Paroline ad ʒi.

The disease can rarely be cured, but the symptoms can be very much relieved.

### 3. DISEASES OF THE MAXILLARY ANTRUM

**ACUTE NON-SUPPURATIVE INFLAMMATION.**—Infection from the nose is the causative agent in all cases.

**Symptoms.** Pain in the affected antrum, neuralgia, and a feeling of tension are usually complained of. In some cases sudden gushes of clear fluid from the nose occur, with a temporary relief from the symptoms.

**Diagnosis.**—The nasal mucous membrane is seen to be acutely inflamed. Transillumination may in some cases assist the diagnosis, but is most unreliable.

**Treatment.**—Usually if the antrum is punctured under the middle of the inferior turbinal bone and the fluid drawn off, the condition rapidly settles down. Repeated punctures are sometimes necessary. A spray of 5 per cent. methyl salicylate in paroline should be used every day until the inflamed condition of the mucous membrane has resolved.

**ACUTE SUPPURATION.**—The infection is usually from the nose or from a tooth.

**Symptoms.**—The onset is sudden; there is acute pain in the antrum, with headache and neuralgia, tenderness on pressure over the canine fossa, nasal obstruction, and a muco-

## SINUSES, ACCESSORY AIR, DISEASES OF

purulent or purulent discharge from the nose. There may be oedema and redness of the tissues externally. The temperature is usually raised.

**Diagnosis.**—The nasal mucous membrane is always acutely inflamed, and if a discharge is present it will be seen escaping from the antrum under the anterior end of the middle turbinal bone. In some cases, if the pus is wiped away and the patient tilts the head well over towards the opposite side, it will appear again in large quantities.

In some very acute cases actual bulging-in of the outer wall of the nose takes place.

**Treatment.**—After the middle third of the inferior turbinal bone has been removed, the antrum should be punctured with a Lake's mastoid gouge so as to establish free drainage; failure to provide free drainage is the reason why so many cases become chronic.

After-treatment consists in washing out the antrum with normal saline solution daily until the condition has completely settled down.

Diseased teeth must be removed, but in no circumstance must the antrum be punctured through a tooth socket, otherwise the disease will certainly become chronic.

When the neuralgia is severe, aspirin usually gives great relief. Most cases react to treatment very rapidly.

**CHRONIC SUPPURATION.**—As in the acute form, this disease originates in infection from the nose or from a tooth or teeth.

**Symptoms.** The symptoms most often complained of are a purulent or muco-purulent nasal discharge, frontal headache, nasal obstruction, pain in the antrum, crust-formation, and sometimes a bad odour.

Aural, pharyngeal, and laryngeal complications are almost always present when the disease has existed for any length of time, and general symptoms, such as indigestion and a feeling of general malaise, may occur.

**Diagnosis.**—Pus will be seen in the position already described in connexion with the acute form. In the early stages there is usually enlargement of the anterior ends of the middle and inferior turbinal bones, but as the disease progresses an atrophic condition often supervenes. In rare cases polypi arising from within the antrum may be seen in the nostril.

**Transillumination test.**—In cases of suppuration the bright crescent of light under the orbit is either dimmed or completely obliterated. The test, taken by itself, is most unreliable, as it is possible to get a bright crescent of light

in an antrum full of pus, and a normal antrum may show no light reflex whatever. The test, therefore, is only of value when its results confirm what has already been ascertained by a careful review of the symptoms and by anterior rhinoscopy.

The practitioner must always make certain that any dental plates have been removed before the transillumination test is applied. When there is any doubt in the diagnosis the antrum should be punctured and washed out; the presence of pus will confirm the diagnosis.

It must be remembered that the antrum is very apt to become infected in cases of frontal and ethmoid sinus suppuration, and therefore the presence or absence of suppuration in these sinuses must be ascertained before any treatment is carried out.

**Treatment.**—The radical operation, combined with the removal of any carious teeth which may be present, is the only cure for the condition when it has been present for a considerable time. When it is of more recent origin the intranasal operation is indicated. After-treatment consists in the use of a saline douche morning and evening, followed by a spray of 5-per-cent. menthol in paroline in cases where there is any crust formation.

**ANTRAL CYSTS.** Some cysts in the antrum are due to degeneration of portions of the lining mucous membrane, but most of them are dental in origin. The symptoms are not usually severe, in most cases only a swelling of the anterior wall of the antrum being complained of. Sometimes a typical "egg-shell" crackling can be obtained on palpation. Complete excision of the cyst must be undertaken in all cases.

### 4. DISEASES OF THE SPHENOIDAL SINUS

**ACUTE SUPPURATION.** This condition is caused by the spread of infective processes into the sinus from the nose or postnasal space.

**Symptoms.**—There is occipital or frontal headache of sudden onset, accompanied by a sense of tension and pain at the back of the orbit. A purulent or muco-purulent postnasal discharge is sometimes present. General symptoms such as raised temperature and severe malaise are usually in evidence.

Thrombosis of the cavernous sinus is very apt to complicate this condition, in which case blindness, fixation of the eyeball, proptosis, chemosis, dilatation of the pupil, accompanied



## SINUSES, ACCESSORY AIR

by intense headache, rigors, and very high temperature, are followed by the onset of symptoms of meningitis; the condition is always fatal. Meningitis may also occur independently of thrombosis of the cavernous sinus.

**Diagnosis.**—By posterior rhinoscopy pus may be seen passing down over the posterior ends of the middle and inferior turbinal bones on the affected side, while by anterior rhinoscopy pus may be found passing down over the body of the middle turbinal bone. In most cases the nasal mucous membrane is acutely inflamed.

**Treatment.**—Inhalations of compound tincture of benzoin and eucalyptus or menthol give great relief. The sinus must be washed out by means of a sphenoidal-sinus catheter at frequent intervals during the first few days. If the symptoms do not react to treatment at once, or if in spite of treatment they continue to increase in severity, free drainage must be established by the removal of as much of the anterior wall of the sinus as possible—a very difficult and dangerous proceeding owing to the close proximity of the cavernous sinus.

Cases uncomplicated by sinus thrombosis or meningitis usually react well to treatment.

**CHRONIC SUPPURATION.**—The chronic arises in the same way as the acute variety.

**Symptoms.**—Pain behind the eyes, and in some cases frontal or occipital headache, are the symptoms usually complained of. Crusting sometimes occurs in the postnasal space owing to the drying of the discharge which is always present in varying degrees; decomposition of the crusts often imparts a foul odour to the breath.

Thrombosis of the cavernous sinus and meningitis are the two complications most to be feared.

Symptoms due to aural, pharyngeal, and laryngeal complications, such as deafness, tinnitus, discharge from the ear, pharyngitis sicca, laryngitis sicca, may occur in the later stages. In these stages also the posterior ethmoidal cells are very apt to become involved.

**Diagnosis.**—Pus is seen in the positions described under Acute Suppuration, and the crusts are often found adhering to the postnasal and pharyngeal mucous membrane. The diagnosis is confirmed by the insertion of a catheter, when, on inflation, pus bubbles out of the sinus. X-ray photographs sometimes assist, but must be interpreted by an expert.

## SKIN, FIBROMATA OF

**Treatment** consists in the daily washing out of the sinus and the use of an oily spray to prevent "crusting." Should resolution not occur, the anterior wall of the sinus must be removed to establish free drainage.

G. N. BIGGS.

## SINUSES, CEREBRAL, THROMBOSIS OF (see CEREBRAL SINUSES, THROMBOSIS OF).

## SIRIASIS (see SUNSTROKE).

**SKIN, FIBROMATA OF.**—Cutaneous fibromata, when multiple, have been called *Molluscum fibrosum*. They constitute an ill-defined group of which many instances are examples of *von Recklinghausen's disease* or neuro-fibromatosis. As originally described, this disease included three elements: (1) neuro-fibromata (fibromata) of the skin, (2) true neuro-fibromata of the peripheral nerves (see NEUROMA), and (3) multiple areas of pigmentation. There is often an associated mental defect of slight or greater degree. Examples are frequently encountered in which one or more of the above elements are wanting.

(1) The fibromata form pedunculated or sessile tumours varying in size from a pea to an orange. When they are of large size the covering skin is coarsened from dilatation of the pilo-sebaceous orifices. As the result of injury, inflammatory reactions may be met with. These tumours can often be invaginated by pressure, a process recalling the reduction of a hernia. Occasionally a tumour assumes gigantic proportions, hanging down in large pendulous masses. Apart from the effects of trauma already alluded to, the disorder is painless, although by appropriate staining methods nerve-fibrils may be demonstrated in the growths. The number of these tumours varies in different cases, and may be considerable.

(2) True neuro-fibromata occur in the course of the peripheral nerves as multiple painful nodules, but are wanting in the majority of cases. (3) The third element, pigmentation, is commonly present, and consists of macules from the dimensions of a freckle to patches the size of the palm. The degree of pigmentation is also variable, shades from light yellow to deep brown being seen.

Strictly speaking, *von Recklinghausen's disease* is a congenital defect, although it may not become manifest until some years after birth. Once it is established, progress is con-





Extensive rodent ulcer in a woman aet. 83.



Rodent ulcer of moderate degree.

PLATE 30.—RODENT ULCER.

## SKIN, MALIGNANT GROWTHS OF

tinuous, new tumours appearing from time to time. Instances are recorded in which spontaneous retrogression has occurred.

**Treatment.**—Apart from surgical removal, treatment is unavailing. If considered desirable, prominent or inconvenient tumours may be thus dealt with. H. MACCORMAC.

### SKIN, MALIGNANT GROWTHS OF.

—In this article cutaneous carcinoma, including rodent ulcer and melanotic carcinoma, will first be considered, and then sarcoma.

#### PRIMARY CANCER OF THE SKIN

Carcinoma of the skin is analogous to cancer elsewhere, except that the tendency to form metastases is less in evidence. The melanotic growths, however, do not conform to this general rule, their course being characterized by rapid and extensive dissemination. The disease may assume various forms, warty, papillomatous, or ulcerative. Crusting and cicatrization are features of the superficial types. It should also be remembered that certain simple conditions such as senile keratosis and chronic ulceration may undergo secondary malignant change.

**Etiology.**—Cancer of the skin is a relatively common condition, and is met with in both sexes in middle life or later, being rare under the age of 30. Two types are recognized, rodent ulcer and squamous-celled carcinoma. The former is most often met with on the face; the latter not infrequently arises upon some pre-existing affection such as lupus vulgaris, senile keratosis, burns—as in kangri cancer—or chronic ulceration. The well-known tendency for X-ray dermatitis to undergo malignant change may be mentioned, although this form of disease should now be but rarely encountered. Cancer of the skin seems to be peculiarly prevalent in some parts of Australia.

**Pathology.**—Rodent ulcer and squamous-celled carcinoma have each characteristic microscopic appearances. It will be remembered that the epidermis comprises a basal layer of small oat-shaped cells, and above this several layers of larger polyhedral cells between which intercellular fibrils can be seen. It is usually held that rodent ulcer arises from these basal cells, and squamous-celled carcinoma from the larger polyhedral cells. In both varieties the epithelium invades the corium, growing down in lobules or becoming detached as separate islands. Some cells may be carried to neigh-

bouring lymphatic glands, forming metastases. Hypertrophy of the sebaceous glands in the region of the growth is especially common in the case of rodent ulcer, while in both forms of growth marginal collections of plasma-cells occur. Cystic degeneration is met with in some forms.

It is usually said that in rodent ulcer the degree of malignancy is less and lymphatic contamination rarer than in squamous-celled carcinoma, but the writer believes that too much stress has been laid upon these differences and that the two types are more closely allied than is generally held. The explanation of the exaggerated view of the malignancy of squamous-celled epithelioma would appear to result from the inclusion under that term of two conditions, squamous epithelioma of the skin proper and squamous epithelioma of mucous membranes, e.g. those of tongue and lip. While the microscopical appearances of these are the same, their clinical course is different; if the purely cutaneous squamous-celled cancers be considered separately, it will be found that they differ but little in their clinical course from rodent ulcer. Nor should the enlargement of lymphatic glands always be accepted as evidence of metastasis, for such enlargement is often inflammatory in character and due to sepsis in the primary growth.

**Symptomatology.**—(1) **Rodent ulcer** (PLATE 30).—This is probably one of the most chronic of all forms of cancer. The upper two-thirds of the face is the area most liable to involvement, although the skin on any part of the body may be affected. Occasionally the growths are multiple, two or more lesions being present. In its earliest form the tumour is represented by a small waxen papule, unusually hard on palpation; or the form of a ring may be assumed. Tiny vessels can frequently be observed crossing over the surface of the growth. In the rare cystic variety translucent areas appear from which fluid may be obtained by puncture. There is also a flat variety resembling a piece of card let into the skin. As the growth advances, different appearances are met with according to whether it extends superficially or deeply. In the former case, a central superficial ulceration occurs, usually with the formation of scar tissue. The characteristic hard rolled edge persists in the form of a circle or segments of circle. Crust-formation is a common feature, and such crusts may completely hide the underlying growth; when, therefore, a

## SKIN, MALIGNANT GROWTHS OF

crust is seen on the face of an elderly person it should be removed and the underlying condition carefully examined. In all cases the extreme hardness of the edge of the growth constitutes an important diagnostic feature. The form that invades the tissues deeply produces considerable excavation and may cause extensive mutilation and destruction. Hardness of the edge is also characteristic of this variety.

(2) **Squamous-celled carcinoma**, though it may occur in apparently normal skin, more commonly develops upon a pre-existing abnormal state such as lupus vulgaris, chronic ulcer, tar or chemical dermatitis, or senile keratosis. Any part of the body may be involved, but the more usual sites are the pinna, neck, face, and back of hand. The growth may be nodular, warty, or ulcerative. As in the case of rodent ulcer, hardness of the edge of the growth is characteristic. The warty form is more common on mucous membranes than on the skin. Glandular metastasis may be met with.

**Diagnosis.** The age of the patient is important in deciding whether a growth is malignant or not. The site, and the characteristic hardness of the edge, must also be considered. Rodent ulcer should be distinguished from *syphilis* and *lupus vulgaris*. In the former disease the destruction is deeper as a rule, and the progress more rapid, and other evidence of infection may be obtained from the Wassermann reaction, from leucoplakia of the tongue, symmetrical enlargement of the epitrochlear glands, and other signs of *syphilis*. *Lupus vulgaris* is of long duration, usually beginning in childhood. The most certain methods of diagnosis rest upon the evidence of microscopical examination; and in any case of doubt a tiny portion of the edge of the growth should be removed for this purpose.

In distinguishing between rodent ulcer and epithelioma, a much more difficult problem is presented. If the squamous-celled cancer of mucous membrane be eliminated, it may be said that epithelioma is very often secondary to some other condition, such as burns, senile keratosis, soot- or paraffin-dermatitis, etc. In all varieties the hardness of the growth is a characteristic feature. The presence of enlarged lymphatic glands does not necessarily prove malignancy, as has been pointed out; hardness of the glands is of more importance. Nevertheless, it is always wise to remove such

glands, since their nature can only accurately be determined on the result of microscopical examination.

**Prognosis.**—If the distinction between cancer of the skin and of the mucous membranes be allowed, then it may be said that in skin cancer the prognosis is fairly good, and this is especially true of rodent ulcer. The earlier treatment is undertaken, the better the chance of cure. Cancer following X-ray dermatitis forms an exception to this statement.

**Treatment.**—In **epithelioma** surgical removal is probably the best method. Any enlarged glands should be dealt with at the same time. In **rodent ulcer** the choice of methods is larger. Here also excision may be undertaken, but other forms of treatment give better cosmetic results. Treatment by radium is usually satisfactory (*see* RADIUM-THERAPY). The growths may also be made to disappear by irradiation with X-rays, doses equal to one or even one and a half pastille being given at intervals of three or more weeks. Precautions should be taken to prevent damage to the skin in the neighbourhood. Freezing with CO<sub>2</sub> snow has many advocates; frequently it entirely removes the smaller types of growth. Consideration of the pathology will show that the application must be deep and of sufficient duration if the whole growth is to be influenced by the freezing. Repetition is usually necessary. Caustics on the whole are unsatisfactory, and may even aggravate the growth from the irritation they produce.

**MELANOTIC CARCINOMA** (*syn.* Nævo-carcinoma; Melanotic Sarcoma).—This is one of the most malignant forms of cancer known. The growth takes origin in a pigmented mole on some part of the body, more commonly on the face or the plantar region; at times it may be very difficult to discover the original site. The disease is a rare one and affects adults, including the aged. The earliest indications are local. The mole becomes painful and reddened, growing slightly, while pigment may be deposited about it. Metastases form with extreme rapidity, numerous sooty nodules appearing underneath the skin at distant parts. As a rule the viscera are extensively affected. The lymphatic glands may also be involved. Treatment is unavailing, once dissemination has occurred. Since the disease arises from an "irritated mole," it is well to remove any lesion of this sort directly it shows signs of activity. Nor should a mole

## SKIN, MALIGNANT GROWTHS OF

be treated by any method which is likely to cause such irritative changes. Some have even gone so far as to advise the removal of any deeply pigmented mole as a prophylactic measure.

A somewhat similar variety of new growth may originate in the choroid of the eye; this, however, is a sarcoma, not a carcinoma.

### SECONDARY CANCER OF THE SKIN

This form of cancer may arise as a result of direct extension or by metastases. Nodules may develop in the skin secondary to carcinoma of the breast or of the viscera. When the primary lesion is evident, diagnosis is simple; but if the nodules are secondary to visceral disease, unless the association be kept in mind mistake may occur. In any dubious case the question can be settled by microscopical examination.

### SARCOMA OF THE SKIN

Cutaneous sarcoma is relatively rare. Like carcinoma, it may be either primary or secondary. The *primary* form is met with as a dark-red or violet tumour or as pale nodules, varying in size from a pea to a walnut. The pigmented forms should be distinguished from melanotic carcinoma, which, it will be remembered, takes its origin in a mole. As in sarcoma in general, the difficulty of distinguishing between new growth and an inflammatory process is often considerable, both clinically and microscopically. *Secondary* sarcoma results from the presence of primary disease in such positions as the testicle or bone. The treatment of all forms is surgical.

**MULTIPLE IDIOPATHIC HÆMORRHAGIC SARCOMA OF KAPOSI.** Though once regarded as a sarcoma, this disease is now known to be inflammatory. It occurs almost entirely in males, beginning on the extremities as flat infiltrations in which dusky tumours develop. The tumours and infiltrations, which are symmetrical and of slow growth, spread from the periphery towards the centre. Necrosis and ulceration are terminal changes. Life may be prolonged for many years. Treatment is unsatisfactory. In some cases arsenic has given favourable results, and applications of X-rays may cause the individual tumours to diminish or disappear.

H. MAC'ORMAC.

### SKIN, SYPHILITIC LESIONS OF (see SYPHILIS).

## SKIN, TUBERCULOSIS OF

**SKIN, TUBERCULOSIS OF.**—The *Bacillus tuberculosis* is the cause of an important group of cutaneous diseases. The organism may reach the integument by—

- (1) Direct inoculation through a breach of the surface or some pre-existing lesion, as in many cases of lupus vulgaris, tuberculous ulcer, and tuberculosis verrucosa.
- (2) Extension from a tuberculous lesion of an adjacent mucous membrane, as in lupus of the nose.
- (3) Invasion of the skin by the breaking down of a tuberculous focus in a gland, bone, or joint, as in scrofuloderma and certain cases of lupus vulgaris.
- (4) Infection of the blood-stream, the bacillus of Koch being derived from some deep-seated focus, as in multiple lupus following the acute specific fevers, miliary tuberculosis, and the tuberculides.
- (5) Infection spreading from an old lesion along the lymphatics—tuberculous lymphangitis.

To classify a cutaneous affection as "tuberculous," three conditions are necessary:

1. The bacillus of Koch must be present in the lesions.
2. Tuberculosis must follow the inoculation of a guinea-pig with the diseased tissue.
3. The injection of Koch's old tuberculin must cause a reaction in the lesion.

In the conditions known as "tuberculides" the patient usually shows evidence of tuberculosis in other organs, but it is exceptional to find the bacillus of Koch in the skin lesions, and, moreover, inoculation of the guinea-pig usually gives negative results, and the reaction to the injection of tuberculin is variable.

### MILIARY TUBERCULOSIS OF THE SKIN

This is a rare condition occurring in children suffering from generalized miliary tuberculosis. The outbreak, which usually lasts from a week to a fortnight, appears to be most common after measles. The lesions are small, acuminate, red papules or papulo-vesicles, rarely pustules, and seldom larger than a hemp-seed. In later stages the papulo-vesicles become scaly, or a minute crust forms. The cutaneous manifestation is part of a general tuberculosis, and death usually occurs from meningitis. Histologically, the papules show giant cells containing Koch's bacilli.

## SKIN, TUBERCULOSIS OF

### TUBERCULOSIS CUTIS ORIFICIALIS

An acute variety of tuberculous ulcer occurring at the orifices. It is due to direct inoculation of muco-cutaneous surfaces with virulent tubercle bacilli derived from "open" tuberculosis of the viscera.

The primary lesion is a round papule which rapidly breaks down into a shallow ulcer with undermined edges. The ulcer rarely exceeds a threepenny-piece in size. It is exceedingly painful and tender, and in the discharge tubercle bacilli are usually found in large numbers. I have met with four types of case:

1. On the lower lip at the angle of the mouth, in cases of pulmonary tuberculosis. It is probably due to the direct inoculation of infected sputa. A similar ulcer may be seen at the nostril from infection by sputa on the handkerchief.

2. At the anus, occurring in association with fistula in advanced tuberculosis when the intestinal tract is involved.

3. At the meatus urinarius in the male, in association with tuberculosis of the urinary tract.

4. Around a tracheotomy wound in pulmonary and laryngeal phthisis.

The ulcers rarely extend, and **treatment** is purely palliative. It may be necessary to apply cocaine to the lip lesions, as the pain may prevent the taking of proper nourishment. Short applications of the X-rays are useful, and in some cases painting with iodine (1 in 5) after cocaineization has been of benefit.

### TUBERCULOSIS VERRUCOSA

This condition, also known as "anatomical tubercle," "post-mortem wart," "necrogenic wart," "butcher's tubercle," is a warty condition of the skin due to direct inoculation of the tubercle bacillus.

**Etiology and pathology.** It is most commonly seen in those in attendance on patients suffering from "open" tuberculous disease; e.g. I have known it follow the washing of handkerchiefs infected with tuberculous sputa. Pathologists, post-mortem attendants, veterinary surgeons, butchers and others who handle carcasses infected with tuberculosis also suffer, the lesions being known as "necrogenic warts." Histologically, the lesions consist of characteristic tubercles containing Koch's bacilli in their giant cells. Miliary abscesses occur in the true skin, and the horny layer is much thickened. Koch's bacillus is found in the pus and may be demonstrated in

sections of the lesions. Inoculation of a guinea-pig gives positive results.

**Symptomatology.**—The fingers, thumb, and back of the hand are the parts most frequently affected. The primary lesion is a small red swelling upon which a pustule develops. The area becomes indurated and slowly extends, finally forming a nodular warty swelling surrounded by a red zone. In some cases the lesion is a rounded plaque which spreads very slowly and may involve the greater part of a finger or cover the back of the hand. After several months, or even years, tuberculosis verrucosa presents a very characteristic appearance. The centre consists of a depressed cicatrix, around which is a ring of small warty nodules usually covered with a crust the colour of dried putty, and outside this again is a zone of erythema. In all cases of warty tuberculosis the lymphatic glands are infected early. Visceral disease may follow.

**Diagnosis** is usually not difficult. The long history and steady extension, with a zone of inflammatory erythema around the lesions, serve to differentiate tuberculosis verrucosa from common warts. An *extragenital chancre* is more rapid in its evolution, and spirochaetes can be demonstrated by dark-background illumination.

**Treatment.**—The wart may be excised, but in the chronic and spreading variety the application of strong creosote and salicylic-acid plasters (33.3 per cent. of each), followed by short exposures to the X-rays, has proved satisfactory in many cases under my care.

### SCROFULODERMIA

This name is given to a gummatous type of tuberculosis affecting the deeper layers of the true skin and hypoderm, which later develops into a chronic type of ulceration.

**Etiology.**—Scrofuloderma occurs usually in poorly nourished children and young adults as a sequel to tuberculosis of the glands, bones, and joints.

**Pathology.**—The lesions consist of a number of caseating tubercles which may be enclosed in fibrous tissues but are more frequently diffuse. Tubercle bacilli are usually present, and guinea-pig inoculation gives positive results.

**Symptomatology.**—Scrofuloderma commonly occurs in the neck, groins, or on the limbs, but may involve the face and trunk. In the form known as tuberculous lymphangitis, which usually follows verrucose tuber-

## SKIN, TUBERCULOSIS OF

culosis on the foot or hand, the lesions are situated at intervals along lymphatic trunks, e.g. along the short saphena vein at the back of the leg, or along the forearm.

The first stage is a painless swelling in the subcutaneous tissue or dermis. The tumour softens in the centre and the surface becomes purple or livid. Ulceration is usual. The ulcer is irregular in outline and its edge is thin, bluish, and undermined; the base is composed of pale granulations covered with greyish-yellow debris. Fistulous tracks run under the skin, and sometimes several orifices form. The discharge is usually thin and serous or seropurulent, but may be mixed with blood. In the extremities, tendon-sheaths, bones, and joints may be involved. Sometimes slowly-spreading indolent ulcers develop which may remain open and discharging for years. On healing, the irregular surface of the scar is often very disfiguring. The scars frequently adhere to the deep tissues, and may impair movement. In the lymphatic type the limb is often greatly swollen from lymphatic obstruction, the skin and subcutaneous tissue being in a condition of solid oedema—a form of pseudo- elephantiasis.

**Diagnosis.**—Scrofuloderma following previous tuberculosis of the glands, bones, or joints is easily recognized. It differs from syphilitic *gumma* in its slower evolution and in the undermined character of the edge of the ulcer. Those rare conditions *sporotrichosis* and *blastomycosis* are diagnosed by cultures of *sporotrichium* and *blastomyces* made from the pus. Bazin's *erythema induratum*, a tuberculide, is usually symmetrical, and affects the lower limbs in young girls (*see* p. 372).

**Treatment** is usually surgical, the skin lesions being removed at the time the affected glands, bones, or joints are dealt with. When the ulceration is confined to the skin it may be excised if not too extensive, but great benefit often follows treatment by minute doses of tuberculin, beginning with  $\frac{1}{10000}$  to  $\frac{1}{1000}$  mg. Short exposures under the X-rays (half-pastille doses) at intervals of a fortnight, and dressing of the ulcer with iodoform or a similar antiseptic, are often of great service in healing the cutaneous ulcers. Good food and prolonged residence at the seaside are of the greatest value.

### LUPUS VULGARIS

Lupus vulgaris is a granuloma due to the tubercle bacillus, affecting the skin and adjacent mucous membranes. It spreads slowly by

the invasion of surrounding normal skin and by the formation of fresh foci. The tissues involved are destroyed either by ulceration or by cicatrization.

**Etiology and pathology.**—The majority of the patients are attacked in childhood or adolescence. In at least one-half the cases the disease begins before the tenth year. In a very small proportion its onset is delayed to an advanced age. Females are more often attacked than males; in my clinic the proportions are 70 to 30. Lupus is much more common in the children of the poor and ill fed, but is not confined to the indigent classes. The most destructive types of the disease are seen in those who live under bad hygienic conditions. There is a history of tuberculosis in the family in 40 per cent. of the cases, and occasionally one meets with cases in which there is reason to suspect direct inoculation from the sputa of consumptives living in the house. It is, however, rare to find more than one member of a family suffering from lupus vulgaris. It is much more common in cold, damp climates than in tropical or subtropical regions.

The infection may occur by direct inoculation as in tattooing, piercing of the lobule of the ear for earrings, morphia-syringe punctures, and occasionally in wounds. Inoculation of the nostrils and of impetigo lesions elsewhere by infected fingers is probably a common cause. In other cases the infection spreads from a deep focus, e.g. tuberculous glands, bones, etc., as in scrofuloderma, and cases of scrofuloderma are sometimes complicated by lupus. In one type where numerous small lesions in large numbers occur almost simultaneously after an acute specific fever, particularly after measles, it is believed that the fever causes a distant focus, for instance a gland affected with tubercle, to soften and thus permit the escape of bacilli into the blood-stream. This type is classed as lupus post-exanthematicus.

Histologically, the lupus-nodule is a granuloma consisting of masses of round, nucleated cells in a fine reticulum of connective tissue; giant cells and plasma cells are common. The bacillus of Koch is found, but in very small numbers. The lupus-nodule does not caseate but undergoes fatty degeneration. The glands of the skin, the hair-follicles, and ultimately the whole structure of the integument is replaced by scar tissue. The epidermis may be unaffected, but in old cases it may be atrophic or undergo great thickening—hyperkeratosis.



## SKIN, TUBERCULOSIS OF

The injection of Koch's old tuberculin causes a local and a general reaction.

According to Stanley Griffith, both human and bovine types of bacillus are present, but in the majority of the cases submitted to him from my clinic the material gave modified forms of the two types.

**Symptomatology** (Plate 31).—The characteristic lesions at the onset are flat papules, from a pin-head to a lentil-seed in size, dark-red or brownish-yellow. They are best seen under glass pressure which removes the surrounding hyperæmia and displays the translucent yellowish-brown appearance which has been likened to "apple jelly." The papules are soft, and are easily shelled out by a curette. They appear singly or in groups, and plaques or patches are formed by their coalescence. The surface may be smooth or covered with fine scale, and in some instances by a thickened horny layer (*lupus verrucosus*). In many instances the patches are flat, while in others they are distinctly raised above the surrounding skin.

The patches may undergo cicatricial change in the centre, while fresh nodules appear at the margin and the diseased area slowly increases; the margin may thus have a serpiginous or rounded outline (Plate 31, Fig. 2). Even in the central scarred area it is common for fresh nodules to appear from time to time. A second change is the breaking down of the patch into an ulcer. The ulcers are irregular in contour and shallow. There is little secretion, but hæmorrhage from the surface is fairly common. Hypertrophic granulations may appear at the edge of the ulcerated area.

A characteristic feature of *lupus vulgaris* is its slow progress. Nodules and plaques may remain stationary for years and may then undergo the retrogressive changes mentioned, or they may slowly extend by peripheral extension or by the formation of fresh discrete nodules which ultimately coalesce with the original patches.

**Mucous membranes.**—The mucous membranes were attacked in 43 per cent. of my cases, and in many instances the primary lesion occurred on them. The inferior meatus of the nose is often the site of infection. From the nose the lupus may extend along the nasal duct, causing epiphora. From the nasal duct it may spread to the lachrymal sac and conjunctiva. From the nasal cavity the disease may extend backwards to the naso-pharynx and through the anterior palatine foramen to the hard palate

just behind the incisor teeth. Both the hard and the soft palate may be attacked, but I have never seen a perforation of the bony palate by lupus, as in syphilis.

The nose is the most frequent site of infection; next in frequency come the lips and buccal mucosa and the palate.

The earliest mucous-membrane lesion is a raised patch with a granular surface. On this surface small superficial ulcers develop. In the nasal cavity the ulcers are commonly covered with adherent crusts. When the gums are involved there is considerable swelling and congestion, followed by ulceration which spreads along and exposes the roots of the teeth.

**Complications.**—Erysipelas is not uncommon, and I have seen cases in which an attack of erysipelas has been followed by the disappearance of the whole or part of a lupous area.

Visceral tuberculosis also occurs, and may lead to a fatal issue; it is most common when the naso-pharynx and larynx are involved. Tuberculosis of the bones and joints also occurs in association with lupus.

Carcinoma develops in cases of lupus of long duration. It appears in the cicatricial areas, and also in chronic cases of "red" lupus. Prolonged X-ray treatment of lupus tends to the production of carcinoma. The cancer is usually of the squamous-celled type, but glandular involvement is somewhat rare.

**Diagnosis.**—Lupus has to be distinguished from *scrofuloderma*, which arises in the same class of patient, and, as already mentioned, the two types of tuberculosis of the skin may coexist. The lesions of *scrofuloderma* are gummatous formations adjacent to caseating glands or other tuberculous foci. There are no apple-jelly-like nodules. *Tuberculous ulcers of the orifices* are more acute than lupus. They are always associated with "open" tubercle of the viscera. *Tuberculosis verrucosa* occurs in the extremities, often with a distinct history of infection, and the glands are involved early. *Lupus erythematosus* is nearly always symmetrical, and commonly affects the nose, cheeks, and auricles. There are no apple-jelly nodules.

*Syphilis* differs from lupus especially in the rapidity with which it develops. Lupus takes years to cause the destruction which may be caused by syphilis in a few weeks or months. If the nose or palate is involved, necrosis of bone indicates syphilis. The syphilitic lesions of the nodular or gummatous type show no



PLATE 31.—LUPUS VULGARIS.



## SKIN, TUBERCULOSIS OF

apple-jelly nodules, and the ulcers have steep sides and appear as if "punched out." In any doubtful case the Wassermann test should be used.

In the nodular type of *leprosy* the lesions are large and more raised than the nodules of *lupus*; they are of a dull earthy colour, and glass pressure shows no apple-jelly nodules. In a doubtful case a portion of the tissue should be excised and stained for bacilli. Hansen's bacillus is found in large numbers in the lesions of *leprosy*.

The organisms of *blastomycosis*, *sporotrichosis*, and *actinomycosis* are usually demonstrated without difficulty in their respective diseases, and should be sought for in doubtful cases. In some chronic forms of *sycosis* there may be a difficulty, but the lesions of *sycosis* are pustular from the onset, and pustules centred by a hair can usually be seen at the margin of the affected area.

**Treatment.**—General treatment consists in the administration of good food, and particularly fat-foods, milk, cream, and cod-liver oil. Whenever possible the patient should live an out-of-door life, and a sojourn at the sea is of service. I have not seen benefit from the use of tuberculin in *lupus vulgaris*, and after a long trial have ceased to recommend it.

The local treatment is of great importance. In small areas on parts where the character of the cicatrix is of little moment, e.g. the limbs, excision with a wide margin of healthy skin gives excellent results. Large areas on the limbs often do well with repeated applications of a strong plaster of creosote and salicylic acid (33 per cent. of each). The plaster is applied for forty-eight hours at a time, and when the surface has been thus treated several times the softer *lupus*-nodules often slough out, leaving small cavities which heal up under an antiseptic dressing or after a short exposure ( $\frac{1}{2}$  B dose) of X-rays. I have seen immediate improvement follow the use of Dr. Ellis's brass preparations, but the ultimate results have been unsatisfactory, and I cannot recommend them as curative. An ointment containing pyrogallic acid, ichthyol, and salicylic acid, 40 gr. of each to the ounce, is of service in getting rid of warty masses of epidermis over a chronic *lupus*-patch. For superficially ulcerated areas short exposures to the X-rays often have a rapidly beneficial effect, but relapses are common. When the ulceration affects the nares it is important to make a thorough examination of the nasal cavity, and any *lupus*-

## SKULL, ABNORMALITIES OF

areas there must be dealt with by the curette and cautery. When the mucous lesions are in the mouth, on the palate or lips, I find painting with a solution of iodine, 20 per cent., of service.

In practice the most troublesome lesions to remove are the dry, brownish-red nodules in masses or plaques, so-called *dry lupus* or *lupus non-exedens*. In cases of this type in which the face is involved and the areas are not large, nothing produces such an excellent cosmetic result as the Finsen light treatment. It is tedious and expensive, but in young subjects affected with *lupus* of the face, in whom the cosmetic result is of great importance, the time and trouble involved in the treatment are well repaid by the soft, supple scar. I cannot too strongly urge the importance of recognizing early *lupus* in young subjects, and resorting to Finsen light if the lesions are of the dry type.

Adamson has recently shown some excellent results in the treatment of *lupus* by liquid acid nitrate of mercury.

Finally, I must protest against the persistent use of the X-rays in the treatment of dry *lupus*. Permanent results are rarely obtained, except at the expense of an X-ray dermatitis, with the great risk of cancer developing on the cicatrix. If the rays are used at all they should be applied occasionally in stimulant doses for the treating of *lupus* ulceration, but never in the dry nodular type of the disease. Radium, again, has little influence upon *lupus* of the dry type.

J. H. SEQUEIRA.

## SKIN-GRAFTING (see WOUNDS, TREATMENT OF).

## SKULL, ABNORMALITIES IN SHAPE OF.

—(1) **Asymmetry**, one side being flatter than the other, may result from one-sided compression in utero, or may follow difficult labour. Constant lying on one side in early life, defect of one cerebral hemisphere, premature unilateral closure of sutures, cerebral tumours in infancy, cerebral atrophy and torticollis with unilateral atrophy of face, are other causes. The asymmetry due to moulding, whether before or after birth, often disappears during the first few weeks or months. More circumscribed local bulging or flattening of the skull is often met with from injury at birth, and shows a like tendency to improvement or correction.

(2) **Microcephaly**.—Two varieties are re-

## SKULL, ABNORMALITIES IN SHAPE OF

cognizable. In one the head is round and short from before backwards, retaining, though small in circumference, a shape not far removed from that of normal brachycephaly. In the other the sagittal suture is ridged, and towards this ridge the forehead and sides slope like the sides of a roof. (Fig. 90.) The latter form is accompanied by idiocy, and is thought to be due to premature synostosis of sutures over the undeveloped brain; in the former some mental



Fig. 90.—Microcephaly. (Photo: Dr. H. P. Paterson.)

deficiency is usual, though it is less distinct in type and seldom so profound.

(3) **Macrocephaly.**—Three forms of general enlargement of the skull are readily recognizable:—

(a) *Enlargement of the vault*, which begins at the supraorbital ridges, at the level of which the greatest circumference is found. The skull is resistant, massive, and not yielding, and the fontanelle is flat. Amentia with hypertrophic cerebral sclerosis (macrocephalic amentia) co-exists.

(b) *Hydrocephalic enlargement*, in which the greatest circumference is higher up, giving a top-heavy appearance. The fontanelle is open and bulging and the sutures gape. The bone is thin and yields to the fingers in the neighbourhood of fontanelles and sutures. (See HYDROCEPHALUS.)

(c) The “*hot-cross bun*” head of rickets (q.v.). A similarly shaped skull may be due to congenital syphilis (Parrot’s nodes). It is found earlier in life—during the first month—and differs in that the fontanelle is small and the bones are hard, although it may coexist with small central areas of osteoporosis (cranio-tabes).

(4) In **cretins** the top of the head is flat, the vault being wide and low; in **mongolian imbeciles** the flatness is in the occipital region, the whole cranium being so extremely brachycephalic that the mouth cannot easily accommodate the tongue. (See CRETINISM.)

(5) **Scaphocephaly** (boat-shaped cranium).—This defect is ascribed to premature ossification of the sagittal suture, which projects like the keel of a boat. Lateral expansion of the skull with the developing brain is unable to take place, and the skull in consequence lengthens antero-posteriorly but remains narrow from side to side, producing a very characteristic malformation. Though often associated with backwardness, no degree of amentia necessarily coexists.

(6) **Oxycephaly** (acrocephaly; tower or steeple head).—The vertex towers upwards, especially anteriorly, and the forehead slopes gradually up to the vertex. The superciliary ridges are smoothed out, whilst a prominence can often be felt in the region of the bregma. The same steep ascent is noticeable on the lateral aspects of the cranium, the temporal ridges are similarly ill-defined, and the ears appear to be placed at a lower level than normal. Exophthalmos is present in well-marked cases, and divergent squint and nystagmus are common. Depression of the malar and supramaxillary regions is frequent, the palate being short and highly arched. The appearance of the mouth is reminiscent of severe adenoids. This curious development of cranial bones and characteristic facies is accompanied by serious defect in vision, sometimes by complete blindness, due to optic atrophy, postneuritic in form. Smell, too, is often completely lost. A prominent symptom is headache, and vertigo and fits have been recorded. Oxycephaly is compatible with unimpaired intelligence. Other malformations may accompany it, among them being imperfect extension of the elbow-joints, and flattening, thickening, and curving outwards of the

## SLEEP, DISTURBANCES OF

first phalanx of each thumb. The condition may be congenital, may start within the first few months of life, or may be deferred for a few years. It is ascribed to premature synostosis of the sutures of the vault, especially of the coronal and sagittal. Probably the cranial changes should be regarded as only part of a more extensive osseous dystrophy.

(7) **Prominent temporal bosses** may occur in osteogenesis imperfecta (q.v.).

FREDERICK LANGMEAD.

### SKULL, INJURIES TO (see HEAD INJURIES).

**SLEEP, DISTURBANCES OF.**—It is difficult to give a concise and accurate definition of sleep, since on the one hand it is closely allied to various pathological conditions, as stupor and coma, and on the other to the physiological hibernation into which some animals fall.

Nor can we give a wholly satisfactory explanation of its nature. None of the many hypotheses that have been put forward is sufficient. It is true that the accumulation of waste products which accompanies fatigue often coincides with somnolence, but there is no convincing evidence that it is the cause of somnolence. The occurrence of a hypnotic toxin is purely hypothetical. And the fall of blood-pressure and the cerebral anæmia that occur during sleep are probably only concomitant phenomena. A retraction of the terminal processes on the dendrites of the nerve-cells, which Duval has described, has not been confirmed by later investigators. Sleep may be more reasonably regarded as a manifestation of that period of rest required by all highly developed cells, which is most urgent in the case of the highly differentiated and complex units of the cerebral cortex.

There are various factors, both physical and mental, which favour the onset of sleep. We all know that fatigue of mind or body induces drowsiness, though severe exhaustion may lead to a sleepless night. Our surroundings have also much influence; monotony, or a stuffy room, may send us to sleep before our ordinary bedtime hour, while interesting or exciting company may keep us awake. The tiring or failure of attention that accompanies sleepiness may be a result of it, but it is often an important cause; we are apt to doze during a dull or abstruse lecture or sermon which fails to stimulate or exhausts our attention. Physi-

cal discomfort and pain are frequent sources of sleeplessness, and disturbing thoughts, dreads, and anxieties may keep us awake as effectively as severe pain.

The amount of sleep that is necessary varies in different individuals; some find five or six hours a night sufficient, others require eight or ten. More is necessary in early life, less in old age. Most men acquire a habit of sleep at fixed hours, and fail to obtain adequate rest if this habit is disturbed; even those who have for years worked day and night alternately for certain periods are often unable to adapt themselves to the sudden changes. Sleep is, as a rule, deeper during the early hours of the night than towards morning.

**Insomnia.**—This term, as usually employed, denotes sleeplessness, or lack of sufficient sleep. It may take various forms. Frequently the patient is unable to drop off to sleep within a reasonable time after retiring to bed, and lies for hours awake and conscious of all that is happening around him. This may be due to physical pain or discomfort, or to psychical disturbances, worries, distressing thoughts and anxieties. It is often the result of the action of some poison on the brain, or of a disturbance of the cerebral circulation; it is frequent in all infective illnesses, in nephritis and uræmia, and in poisoning by such substances as atropine, nicotine, tea, and coffee. It occurs, too, in drug-takers, especially in morphomaniacs, and is common in some of the psychoses, as melancholia, and in degenerative forms of cerebral disease; it is sometimes an early symptom in general paralysis. But neurasthenia is the condition to which it is most commonly due; here it may be owing to any of the psychical disturbances of this disease, or it may be that on the cessation of the activities of the day and the absence of external stimuli attention is absorbed by slight visceral changes, as the beating of the heart or movements of the intestines, which loom so large before the nervous patient that they forbid rest. But in neurasthenia it is often the dread of sleeplessness that is its cause; the patient acquires an actual *phobie d'insomnie* and tosses about in a restless certainty that sleep cannot come. Insomnia may also be associated with the nervous symptoms that appear in neurotic women at the menopause and after childbirth.

In another form of insomnia the patient gets to sleep more or less easily, but wakes up at an early hour and is unable to doze again.

## SLEEP, DISTURBANCES OF

Sometimes he lies in a half-conscious state but aware of the passing of time and of every perceptible sound and change around him, sometimes wide awake. This type is particularly common in cerebral arterio-sclerosis and in patients with high blood-pressure and diseased arteries. It is met with in cardiac disease, especially when failure of compensation appears. It occurs also in some of the psychoneuroses, as neurasthenia and hysteria, and in chronic intoxications due to renal, intestinal, and other diseases. It is not infrequent on a change in the habits and hours of sleep, and particularly in those who sleep only during the day.

A third variety of insomnia is characterized by frequent interruptions of sleep during the night. The waking may be due to nightmares or distressing dreams: frights and starts—*pavor nocturnus*—are frequently the cause in early life, especially in children with adenoids and intestinal worms. Pain and physical discomfort, debilitating illnesses or a disturbing environment, are often the cause, or they may be the origin, of broken sleep. Occasionally a patient with Bright's disease or diabetes, who is wakened up by the necessity of emptying his bladder several times during the night and cannot get to sleep again at once, complains of insomnia. Interrupted sleep is also common in neurasthenia.

In a fourth form of disturbance of sleep which may be classed with insomnia, it is the quality rather than the quantity of sleep that is deficient. The patient complains that, though he lies unconscious of his surroundings during the night, he wakes up in the morning tired and unrefreshed. This may be so in patients subject to any form of chronic poisoning, as alcohol or intestinal intoxication, but it is most frequent in neurasthenia and other depressed states.

**Treatment of insomnia.**—In the first place every effort must be made to discover and remove the cause. When it is due to peripheral irritation or pain, or to disease of the heart, kidneys, vessels, or abdominal organs, it is these primary conditions that require attention first. Mental causes should also be searched for; absence from business when it is the source of worry, or the solution of an anxiety or dread, frequently enables the patient to sleep comfortably again (*see PSYCHOTHERAPY*). Hypnotism is frequently successful, especially in patients who have developed a habit of insomnia. It is also extremely

important to make the opportunities for sleep as favourable as possible; the bedroom should be quiet and well ventilated, the bedclothes light but warm, and a hard bed is preferable to a soft one. The patient should not retire with a loaded stomach, or for at least two hours after a heavy meal, but a biscuit or a few mouthfuls of digestible food at bedtime, with a glass of hot milk, often help to induce sleep. Some persons find a small amount of alcohol beneficial, but it is not an advisable remedy in cases of chronic sleeplessness, and if taken near bedtime it tends to keep some patients awake. Stout or a heavy beer, or a glass of burgundy, can be successfully resorted to in suitable cases. Persons who wake up during the night can often get to sleep again quickly if supplied with a hot drink, which may be kept by the bedside in a thermos flask, and a biscuit or a slice of bread.

It is a golden rule not to resort to hypnotic drugs if their use can be avoided, but sedatives, such as the bromides, are safe and useful. The best method of administering them is to give a moderate dose after the evening meal and a full dose at bedtime. In massage and hydrotherapeutic measures, as hot baths, packs and compresses, we have extremely valuable means of dealing with sleeplessness. One of the surest methods in many cases is to order the patient to lie in a hot bath for twenty to thirty minutes, and then, after being dried and rubbed down vigorously with a coarse towel, to have general massage, which should be very gently done as drowsiness comes on.

In some cases the use of hypnotic drugs cannot be avoided. They are particularly necessary when a habit of sleeplessness has developed; then, if the patient can be made to sleep every night for a week or so and be placed in proper hygienic surroundings, natural sleep may return. Morphia and the opiates should be avoided if possible, as increasing doses are required and a drug habit may easily develop. They are necessary only when lack of sleep is due to pain and in melancholia. There are so many hypnotic drugs on the market, and it is so often a matter of finding the one which suits the patient best, that the choice must be left to the individual medical attendant. Mention may be made of trional, veronal, medinal, hedonal, and luminal, which are relatively safe preparations when carefully employed. Paraldehyde is often valuable, though its taste is objectionable. It is advisable to vary the drug that is employed,

## SMALLPOX

or to alternate it with another. It is frequently better to give the dose in two parts, one during the evening and the second portion as the patient goes to bed.

A complete rest from business for a time is usually advisable, and a change of surroundings is often successful in relieving insomnia, but the place where the patient is sent should be carefully selected; some sleep better by the seaside, and others in the country. High altitudes should be avoided as a rule.

**Pathological somnolence** is much rarer than insomnia. In old age sleep is occasionally more prolonged, more frequent, and more profound than normal; and drowsiness, generally associated with mental dullness when awake, is common in many diseases of the brain, particularly in those that raise intracranial pressure, as cerebral tumours and abscesses. Abnormal somnolence is sometimes a feature of general paralysis, and it is one of the most prominent of the symptoms of trypanosomiasis (sleeping sickness). Drowsiness and prolonged deep sleep are so common in epidemic encephalitis as to give that disease the alternative title, lethargic encephalitis.

**Narcolepsy** is a rare neurosis characterized by a sudden and irresistible desire to sleep which recurs at irregular intervals. The sleep, which is very profound, may be of short duration, or may last for days.

GORDON HOLMES.

**SLEEPING SICKNESS** (*see* TRYPANOSOMIASIS).

**SMALLPOX** (*syn.* Variola).—An acute contagious febrile disease characterized by an eruption which, commencing as macules, passes through the successive stages of papules, vesicles, pustules and scabs.

**Etiology.**—The distribution of smallpox is worldwide. It is not only a disease of temperate climates like scarlet fever and measles, but is also prevalent, and accompanied by a higher mortality, in the tropics. All ages are susceptible, congenital immunity being less frequent than in any of the other acute infectious diseases. Even the *fœtus in utero* may be attacked. The comparative rarity of smallpox in children is due to the recent influence of infantile vaccination. The prevalence of the disease is most pronounced during the first forty years of life, but in large epidemics it is not uncommon even in extreme age. Sex has no influence on susceptibility apart

from the fact that pregnant and puerperal women are predisposed to the disease, and especially to a malignant type.

Outbreaks of smallpox may occur at any season of the year, but epidemics in temperate climates usually begin towards the end of autumn or in the early spring.

One attack, as a rule, confers immunity for the rest of life, but a second attack is by no means unknown, though probably in several of the cases reported, one of the two attacks was only varicella. The second attack is usually milder than the first.

The smallpox patient is contagious at all stages of the disease, the infectivity being greatest in the vesicular, pustular and scabbing stages, and least in the periods of invasion and incubation. The date at which the patient ceases to be contagious varies with the severity of the disease from one or two weeks to several months. In practice it is advisable not to release a smallpox patient from isolation until all the scabs have separated and the ulcers have healed.

The contagion is transmitted to the healthy by the smallpox patient, by the body after death, by healthy third persons, and by fomites, such as infected clothing, and letters and parcels sent by post. Transmission by aerial convection, especially in the neighbourhood of smallpox hospitals, has been illustrated in several epidemics, notably in the case of the Fulham Smallpox Hospital in 1883, when the incidence of the disease in the area surrounding the hospital was remarkably high, and again in the epidemic of 1901-2, when there was an unusually heavy incidence of smallpox at Purfleet, situated opposite the hospital ships moored in the Thames. On the other hand, the theory of aerial convection has been rejected by some writers who attribute the spread of the disease in the neighbourhood of hospitals to flies and other insects, to surreptitious visits paid to members of the staff and patients, and to inadequate disinfection by the staff on leaving the hospital.

The contagion probably gains entrance to the body by the mouth, nose, and respiratory tract, and possibly through the unbroken skin.

**Bacteriology.**—The fluid obtained from the vesicles or early pustules is sterile on ordinary media, and it is not until the seventh or eighth day of the eruption that micro-organisms, mainly streptococci, can be grown from the lesions. Streptococci have also been found in the blood of hæmorrhagic cases.



## SMALLPOX

A protozoon-like body named *Cytoryctes variolæ* has been found in the lesions of stratified epithelium of the skin and mucous membrane of the soft palate, pharynx and œsophagus, up to about the sixth day of the eruption. Though its claim to be the causal micro-organism of smallpox has not been fully established, it has been suggested that variola is caused by the combined asexual and sexual life of this parasite, and that vaccinia is due to the asexual phase only.

**Morbid anatomy.**—In addition to the changes in the viscera common to all the acute infectious diseases, such as cloudy swelling and fatty degeneration of the heart, liver and kidneys, and more or less extensive broncho-pneumonia, which are found in the majority of necropsies on smallpox cases, the eruption will be seen in the form of erosions or ulcers on the mucous membrane of the mouth, pharynx, œsophagus, vulva, vagina and lower part of the rectum, as well as in the larynx, trachea, and medium-sized bronchi.

In early cases the spleen is enlarged, soft and diffuent. Small focal necroses may be present in the bone-marrow and testes.

In hæmorrhagic smallpox large and small hæmorrhages are present in almost all the internal organs, as well as in the serous and mucous membranes, loose cellular tissue and skeletal muscles.

Histologically, the specific lesion has been shown to be due to a focal degeneration of stratified epithelium vacuolar in character and accompanied by serous exudation and the formation of a reticulum, the fully developed product being a multilocular pustule.

**Blood.**—During the febrile stage the hæmoglobin and red corpuscles remain practically normal, but postfebrile anæmia is common and poikilocytosis occurs (Councilman). From the vesicular stage onwards the blood shows a moderate leucocytosis (10,000-20,000) due to increase in the number of lymphocytes, which may last as long as three months after the onset of the disease. The neutrophils are diminished.

**Symptomatology.**—The duration of the incubation period is very constant, being nearly always twelve days. The only exceptions are inoculated smallpox, in which it is reduced to seven or eight days, and hæmorrhagic smallpox, in which it may be as short as six or seven days. On the other hand, cases are on record in which it has been prolonged to sixteen or even twenty days. In the great majority of

cases it is not attended by any symptoms at all, but in some instances there may be a complaint of general malaise, headache, backache, and slight gastric disturbance. Catarrhal pharyngitis has been noted in some cases towards the end of this stage (Obermeier).

The **stage of invasion** extends from the outbreak of the first symptoms to the appearance of the characteristic eruption, and lasts as a rule three days, occasionally one day more or less. The symptoms, which may be mild or severe, bear but an imperfect relation to the character of the subsequent attack, very severe initial symptoms being frequently followed by a mild eruptive stage. On the other hand, it is extremely rare for mild symptoms in the prodromal stage to be followed by a severe attack. The disease usually sets in acutely with one or two attacks of shivering, severe headache and backache, giddiness and fever, the temperature rising in a few hours to 104° F. In children generalized convulsions may occur. There is often some pain in the back of the neck and in the muscles, especially those of the lower limbs. Delirium is frequent. The temperature keeps high as a rule during the first three days, and the pulse is correspondingly accelerated. The frequency of respiration is relatively greater than that of the pulse, the rapid and laboured breathing suggesting a commencing pneumonia. There is complete loss of appetite, the tongue is thickly coated, the patient complains of sore throat, which shows a diffuse and patchy redness of the fauces and pharynx, and there is often retching and vomiting. The bowels are usually constipated, but occasionally the stools are loose. Often there is febrile albuminuria at this stage, especially when the attack is at all severe. In women menstruation usually sets in sooner than usual. The spleen is not enlarged at this stage, except in severe cases. On the second or third day *prodromal rashes* are liable to appear. They usually assume two forms, erythematous and hæmorrhagic respectively, which are not infrequently present together. The erythematous form may be either morbilliform or scarlatiniform, and may extend all over the body, usually first appearing on the face. It rapidly fades, and is gone within 12-24 hours, leaving no desquamation. The hæmorrhagic form usually appears earlier than the erythematous, not infrequently on the first day of the prodromal stage. It consists of very small thickly-set hæmorrhages in the upper layer of the skin. Its area of 1 re-

## SMALLPOX

dilection is the lower half of the abdomen and inner surface of the thighs down to the knees, excluding the genitals; it may also extend to the sides of the trunk and axillæ and inner surfaces of both arms. The duration of the hæmorrhagic form is usually longer than that of the erythematous. The areas occupied by the prodromal rash are usually more or less unaffected by the characteristic eruption of smallpox.

**Eruptive stage.**—In the great majority of cases the characteristic eruption of smallpox appears on the third day of disease, but it is sometimes delayed till the fourth day, whilst in modified smallpox it may appear as early as the first or second day. The small red macules of which it at first consists become converted within a few hours into papules that increase in size in the course of the next two days and become surrounded by a narrow erythematous band or areola. On the sixth day of the disease, or the third day of the eruption, a small vesicle appears in the middle of the papule and enlarges during the next two days. On the eighth or ninth day of the disease the individual vesicles have usually reached their greatest development. They are now oval or hemispherical in shape, are multi-locular, and show a central depression or umbilication, which, however, is usually only present in the lesions on the trunk and limbs and absent in those of the face. In most cases the vesicles on the face begin to be transformed into pustules on the fifth day of the eruption, and in the course of the next three days supuration of the pocks becomes generalized. The skin between these lesions becomes oedematous, especially on the face, where the features are obliterated, so that the patient is unrecognizable.

On the twelfth day of the disease the process of scabbing commences, either from rupture of the pustules, which chiefly occurs on the face, or from desiccation of their contents without rupture, which takes place in the lesions on the trunk and limbs, unless they have been ruptured by rubbing or scratching. Desiccation first begins on the face and head, where it is usually completed between the eleventh and fifteenth days, and spreads over the trunk, reaching the lesions on the extremities last of all about the fifteenth day. The swelling and redness of the skin subside, but itching at this time becomes very troublesome.

The scabs, which were at first adherent to the skin, become detached, leaving a pigmented

mark which in course of time becomes pale and, if the supuration has not been deep, disappears altogether. As a rule, however, deep scars are left, especially on the face, giving it a characteristic pock-marked appearance. Separation of the scabs is followed by a branny or powdery desquamation of the skin in the region previously occupied by the pocks.

Loss of hair on the scalp, beard and eyebrows is liable to occur after severe attacks. If the alopecia is merely due to a nutritional disturbance like that following enteric fever or influenza, the hair will grow again, but if the follicles have been destroyed by the pocks, permanent baldness results. The nails of the fingers and toes may also be shed in severe cases.

The eruption does not invade the whole cutaneous surface simultaneously, but during the first two days it develops gradually, appearing first on the face and hairy scalp, then on the chest, upper extremities and back, later on the abdomen, and finally on the lower extremities, the distal parts of which are invaded last of all. In mild cases the whole eruption may be out within twenty-four hours, but in severe cases it may be forty-eight hours or more before the last lesion makes its appearance.

*Distribution of the rash.* The lesions are usually thickest on the face, the back is more affected than the chest, and on the limbs the rash is thicker on the distal than on the proximal parts.

The axillæ, groins, and hypogastrium, which are the areas of predilection for the prodromal rash, enjoy an immunity from the papular eruption.

The rash always shows a tendency to be thickly set in areas where the skin has been subject to mechanical or chemical irritation, e.g. compression by articles of clothing, application of blisters, or a previous skin eruption.

Lesions appear on the various *mucous membranes* about the same time as on the skin. Almost as soon as the vesicles are formed, however, their covering is rubbed off, and ulcers form which may become confluent. The eruption is found in the mouth, nose and throat, and more rarely, and chiefly in confluent attacks, in the larynx, trachea and bronchi. The alimentary canal is usually only affected as far as the middle of the œsophagus and in the lowest part of the rectum. Lesions may also be found in the vulva and lower part of the vagina. The urethra, except the meatus,

## SMALLPOX

almost always escapes. The mucous membrane surrounding the lesions is diffusely swollen and very painful. As a rule, the lesions heal comparatively rapidly on the mucosæ, where they are situated more superficially than those on the skin.

In mild cases the temperature falls to normal very soon after the appearance of the eruption. Patients who have been delirious or sleepless during the stage of invasion recover their senses and enjoy a refreshing sleep. In severe cases, on the other hand, there is only a slight improvement in the general condition and little decline of the fever. As a rule, the temperature is lowest at the beginning of the vesicular stage and then rises during the pustular stage, remaining high for two days or more according to the time taken by the suppurative process. Except in severe attacks accompanied by much suppuration, the temperature is never so high in the pustular stage as in the pre-eruptive period.

**Varieties.**—Four forms of smallpox have been described, viz. (1) confluent, (2) discrete, (3) hæmorrhagic, and (4) modified smallpox, including smallpox without eruption.

1. **Confluent smallpox** (PLATE 32, Fig. 2).—The prodromal symptoms are usually severe. The initial fever is more intense, and there is often delirium, frequently violent. The lesions, as a rule, do not become confluent until the pustular stage, but confluence may occur in the papular stage. Except in very severe cases the confluence of the lesions is confined to the face, arms and hands, the eruption remaining discrete upon the trunk. As a rule, there is a decline of the fever on the appearance of the eruption, but there is not the same remission of temperature and subsidence of the other symptoms as occurs in discrete cases.

During the vesicular period, and still more so during the stage of suppuration, there is considerable œdema of the skin, causing great deformity of the face. During the pustular stage the fever rises to 103° or 104° F. (*secondary fever, or fever of maturation*), delirium is frequent and insomnia the rule. The patient suffers considerable pain from the cutaneous eruption, as well as from the enanthem in the mouth, throat, and larynx. Salivation is well marked, deglutition is difficult, the voice is husky, and there is frequent cough. Fœtor is pronounced during the pustular stage and during desiccation. Death occurs more often at this stage than at any other period.

2. **Discrete smallpox** (PLATE 32, Fig. 1).—In

a typical example the lesions show no tendency to coalesce, but stand apart from one another and can easily be counted. Transitional forms, however, occur in which the lesions are unusually numerous or the eruption is semi-confluent. The prodromal symptoms are, as a rule, not severe, but it is not exceptional for very intense initial symptoms to be followed by a discrete attack. On appearance of the eruption the temperature falls quite to normal, and in mild cases there is little if any secondary fever. Complications are exceptional and the mortality low.

3. **Hæmorrhagic smallpox.**—Two forms of the disease are included under this term—(1) black smallpox, or purpura variolosa, (2) variola hæmorrhagica pustulosa.

The incubation period in *black smallpox* is usually short, and the prodromal period is ushered in with very severe symptoms, especially intense headache and backache, constant retching and vomiting, and prostration. Tonelessness of the facial muscles is a salient sign. The temperature varies; in the more rapidly fatal cases it is apt to be below 100° F. and even subnormal, while in those which survive longer a high temperature is the rule. The mind remains clear until shortly before death.

A characteristic fœtor, quite distinct from that due to decomposition of the cutaneous lesions in confluent smallpox, is given off by the patient, being probably, as Ricketts suggests, an exhalation from the lungs arising from changes in the blood. The liver undergoes a rapid and painless enlargement.

On the second day the initial eruption appears, usually in the form of a deep scarlatiniform erythema, which may be patchy or uniform, confined to the trunk or limbs, or generalized, and is accompanied by a remarkable thickening of the skin. Very soon hæmorrhages are seen on the skin in the form of petechiæ and ecchymoses of varying extent. The face becomes dusky red and swollen, hæmorrhages appear beneath the conjunctivæ and from the other mucous membranes, causing epistaxis, melæna, hæmaturia, hæmoptysis or metrorrhagia. Death may take place within twenty-four hours, but is usually delayed until after the seventh day, by which time definite papules will have made their appearance. A considerable proportion of these cases, however, die before the characteristic eruption of smallpox has had time to develop. Death in many cases is caused by heart failure or œdema of the lungs, and not by the actual loss of blood.

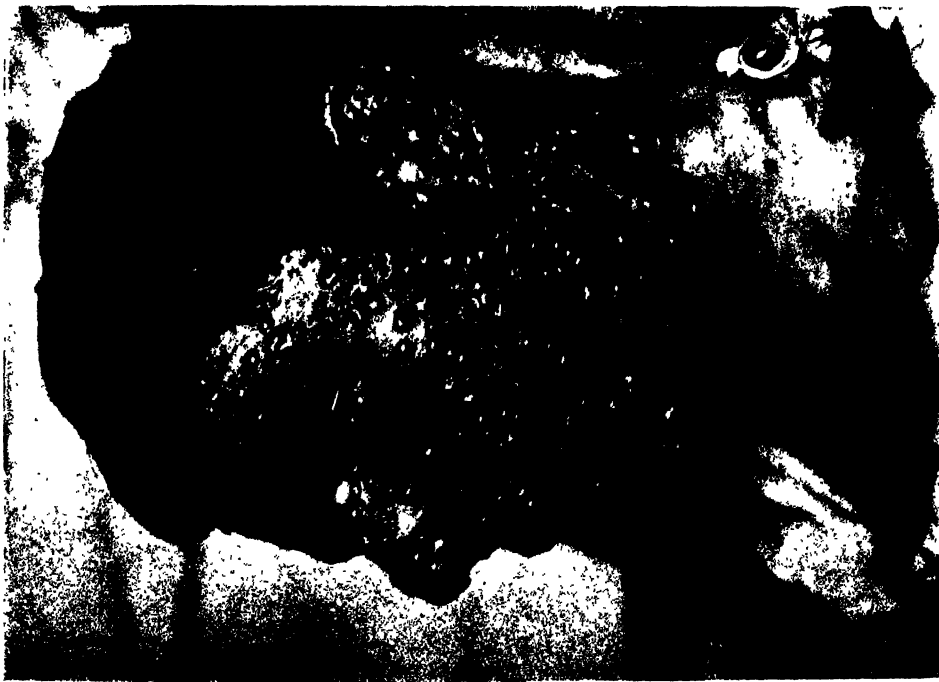


Fig. 1.—Severe discrete rash.



Fig. 2.—Confluent rash.

PLATE 32.—SMALLPOX.



## SMALLPOX

*Variola hæmorrhagica pustulosa* is the more frequent and somewhat less fatal form of hæmorrhagic smallpox. On the third day, the prodromal symptoms being over, the eruption appears, and the subsequent course of the disease varies according as the hæmorrhages appear in the papular, vesicular or pustular stage. The hæmorrhages may be confined to the pocks in special areas of the body, or be generalized. Other hæmorrhages soon appear in the form of petechiæ and ecchymoses between the pocks. Hæmorrhages also occur from the various mucous membranes as in purpura variolosa. In most cases the disease is fatal, and death usually takes place between the seventh and twelfth days, from collapse.

4. **Modified smallpox** is that form of the disease in which, either as the result of a natural resistance to the virus of smallpox, or much more frequently in consequence of immunity acquired by vaccination, there are differences in certain respects from the natural or unmodified form. Its course is shorter and milder, and the individual lesions do not attain their full development. The characteristic eruption may appear on the first or second day of the period of invasion, instead of, as it usually does, on the third. As a rule, the modification of the lesions is not seen until the appearance of the vesicles, which are usually superficial and often uniloculated. In some cases the vesicles are not converted into pustules but into wart-like excrescences, especially on the face, where they persist for a long time after the patient has recovered. The constitutional symptoms are much less severe than in normal smallpox, there is no secondary fever, and complications are rare. Recovery always takes place.

A special form of modified smallpox is what is known as *variola sine exanthemate*, *variola sine variolis*, or smallpox without eruption. After exposure to infection all the initial symptoms of smallpox, including a prodromal rash, develop, and subside on the third day without any subsequent eruption. Such cases, which may be mistaken for influenza, are very apt to occur in recently vaccinated persons shortly after joining the staff of a smallpox hospital.

**Complications. Cutaneous.**—Boils and abscesses are frequent during convalescence, especially after severe attacks. Extensive impetigo may develop during desiccation, and is sometimes followed by local gangrene. Septic rashes in the form of a patchy erythema

may also occur at this stage. Erysipelas is an occasionally fatal complication.

**Respiratory complications** are very frequent. The eruption on the laryngeal mucosa may give rise to considerable dyspnoea. Oedema of the glottis, perichondritis, and ulceration of the laryngeal cartilages may occur. Bronchitis is frequent in otherwise uncomplicated cases. Secondary infection may give rise to bronchopneumonia, hypostatic pneumonia and gangrene of the lung. True lobar pneumonia and pulmonary abscess are very rare. Pleurisy with rapid development of empyema is occasionally seen, especially in confluent cases. The prognosis of these pleural and pulmonary complications is very unfavourable.

**Ocular complications** are fairly common, especially in the pustular stage. Conjunctivitis is frequent and often severe in confluent attacks. Other ocular complications are keratitis, corneal ulcer followed by opacities, iritis giving rise to synechiae, choroiditis, and panophthalmia.

**Aural complications.** Acute otitis media may arise from extension of the catarrhal process from the pharynx through the Eustachian tubes, or may occur independently.

**Adenitis**, especially of the cervical and axillary glands, is common, and often ends in suppuration.

**Bones and joints.**—Serous or sero-purulent arthritis, especially of the large joints, may occur, and sometimes ends in ankylosis. Spread of the inflammation to the articular ends of the bones is rare. Osteomyelitis is exceptional.

**Cardiac complications** are uncommon. They consist of myocarditis, simple or malignant endocarditis, and pericarditis.

**Nephritis** is a rare complication, and is chiefly found in association with pyæmia. Orchitis occurs with varying frequency in different epidemics. Involvement of the other sexual organs is rare.

**Nervous system.**—In addition to headache, insomnia, and severe delirium, cerebral disturbance may be shown by convulsions, which are most liable to occur in children during the prodromal stage. Hemiplegia may result from thrombosis or encephalitis, or be due to cerebral hæmorrhage in hæmorrhagic cases. Aphasia may be associated with hemiplegia or occur independently. Meningitis is a rare complication most frequently seen in children during the period of suppuration and commencing desiccation; it is usually of metastatic origin, but may also be secondary to otitis. Cases

## SMALLPOX

of acute encephalo-myelitis in which the symptoms resemble those of disseminated sclerosis but differ from it by their tendency to recovery, have been reported by several writers. The spinal cord may be involved at any stage of the disease, both in mild and in severe attacks. During the early stage the symptoms are usually vague, consisting merely of backache, retention of urine and pains in the limbs, and subsiding as the eruption appears. Paraplegia of late onset is more serious, as it very rapidly involves all four limbs and proves fatal in a few days. A few cases of anterior poliomyelitis have also been reported. The peripheral nerves are less frequently affected than any other part of the nervous system. A few cases of ulnar and musculo-spiral neuritis and paralysis of the serratus magnus are on record. Neuralgia is frequent in convalescence. Psychoses may occur at any stage, in the form either of acute mania or of melancholia. The prognosis as a rule is good, but permanent dementia may result.

**Smallpox and pregnancy.**—Abortion and premature delivery occur in a high percentage of all severe cases. The fetus is frequently dead at birth, and delivery is followed by severe uterine hæmorrhage and septic inflammation at the placental site.

**Diagnosis.**—In the diagnosis of smallpox, as in most of the other exanthemata, the following points must be borne in mind:—

1. *The history.*—Was the onset sudden or gradual? Were there any special symptoms, e.g. severe backache or headache, noted? Was there a pronounced improvement and fall of temperature in the symptoms at the end of a few days? Did the patient come from an infected home or neighbourhood?

2. *The character of the lesions.*—Are the lesions of the same age, size and shape, or are they polymorphous? In a case of suspected smallpox it is important to determine whether variolous lesions are present in addition to skin lesions which are obviously not variolous.

3. *Areas of predilection.*—The prodromal rashes are mainly found in the abdomino-femoral region, the sides of the thorax and the axillæ, while the characteristic eruption of smallpox is most profuse on the face and distal parts of the extremities, and on the trunk is thicker on the back than on the chest or abdomen.

4. *Evolution of the disease.*—Many diseases can be excluded by their progress. Compared with other pustular lesions, such as acne or

pustular syphilides, smallpox evolves with comparative rapidity, the pustules being soon replaced by scabs, which are not long in separating, leaving pigmented marks.

The distinctive features of the diseases most frequently mistaken for smallpox will now be considered.

*Chickenpox.*—The prodromal symptoms are as a rule slight, or may be entirely lacking, the eruption being the first thing to attract attention. This is, however, by no means always the case. In adults the onset is frequently severe and may closely simulate smallpox. On the other hand, in cases of modified smallpox the initial symptoms may be very slight and the eruption may occur on the first day of the illness. Too much confidence, therefore, should not be placed in the history.

The difference in distribution of the rash is the most important distinction. In chickenpox the abdomen and chest are as thickly covered as the face, or even more so. The lesions are equally numerous on the abdomen and back. On the limbs the eruption is comparatively sparse and thickest at the proximal ends. In smallpox the rash is most profuse on the face, arms and back, and much less abundant on the chest and abdomen. On the limbs the rash is thickest on the distal extremities.

The character of the lesions is also distinctive. In chickenpox the lesions are more superficial, become completely vesicular within a few hours of their first appearance, have a crenated outline which is most pronounced during the desiccation stage, and tend to be heterogeneous. In smallpox, on the other hand, the lesions are more deeply seated, do not become vesicular on the first day of the eruption, are oval in shape, and show a greater uniformity in their age, size and shape in a given area.

The lesions in chickenpox may be very abundant on the face, causing an œdema of the features which is more frequently met with in smallpox. In rare instances the lesions on the face and extremities may be as plentiful or even more plentiful than those on the trunk. A few pocks on the palate and buccal mucosa are extremely frequent in chickenpox, a buccal or faucial enanthem being by no means peculiar to smallpox. The life of an individual lesion in varicella is usually much shorter, but in severe attacks of chickenpox the evolution of the lesions may be somewhat delayed so as to resemble that of smallpox lesions.

## SMALLPOX

**Measles.**—The onset is not so sudden, is preceded by catarrhal signs and Koplik's spots, and is not accompanied by shivering as in smallpox. The individual lesions in measles, on their first appearance, are larger than those of smallpox on the first day of the eruption, which is attended by a fall of temperature, while in measles it continues to rise. Moreover, in smallpox the rash appears first on the face and advances thence over different parts of the body, while in measles the rash develops almost simultaneously on the trunk and face.

**Syphilis** is liable to be mistaken for smallpox, partly on account of the general symptoms—prostration and fever—and partly on account of the eruption. The papular syphilide most frequently, and less often the pustular syphilide, may lead to errors in diagnosis. The distribution of the eruption may resemble that of smallpox, but the diagnosis is usually rendered comparatively easy by the coppery colour of the papules and by the scaly collarette which is never present in smallpox. The characteristic polymorphism of syphilis, and possibly a primary lesion on the genitals or elsewhere, and a Wassermann reaction will be of much help. The progress of the lesions will be decisive. The possibility of the coexistence of syphilis and smallpox, many examples of which occurred in the last London epidemic, should be borne in mind.

**Acne.**—The presence of comedones and characteristic scars, the distribution on the face, shoulders, chest and back, and the slow progress of the eruption indicate acne. As a rule, there is no constitutional disturbance, but some cases may be erroneously regarded as smallpox owing to some digestive derangement. The coexistence of the two diseases is, of course, always possible.

**Prognosis.**—The prognosis depends on the extent of the eruption, and the presence or absence of hæmorrhages. The more scanty the eruption, the more favourable the prognosis. On the other hand, the greater the thickness and extent of the rash, the more likely are dangerous pulmonary or cardiac complications to arise. The outlook in black smallpox is absolutely bad. It is severe, though less unfavourable, in *variola hæmorrhagica pustulosa*. In unvaccinated children under 10 the mortality of smallpox generally is high, and in later life the mortality is greater in females than in males, especially in pregnancy and the puerperium. The mortality in alcoholic subjects is remarkably high.

The situation of the eruption on the mucous membranes is of considerable importance. The presence of a buccal enanthem in infants is serious, as it interferes with their taking nourishment.

The concurrence of mild symptoms in the pre-eruptive stage with an erythematous prodromal rash almost invariably heralds a slight attack. On the other hand, severe symptoms at this stage may be followed by a mild form of the disease. Excruciating pain in the back at the onset should be regarded with apprehension, as it often precedes hæmorrhagic smallpox. Fall of temperature to normal on the appearance of the eruption is of good omen, while an incomplete and delayed fall of the temperature at this stage is of bad prognosis. Severe cerebral symptoms during suppuration are unfavourable.

**Mortality.**—Before the introduction of vaccination the mortality from smallpox was so high that 7 to 12 per cent. of all deaths were due to this cause. In recent times the mortality has varied greatly in different epidemics. In the epidemic of 1870-3 in the M.A.B. hospitals it was as high as 41·8 per cent., owing to the unusually large proportion of malignant and hæmorrhagic cases. Since then the mortality has progressively declined; in the epidemic of 1901-5 it was only 19·3 per cent. among the non-vaccinated. McVail has shown that since the beginning of the twentieth century two different types of smallpox have occurred simultaneously in Great Britain, the one being the European or African type, which originated in North Africa and thence spread to Spain, France, Italy, and London, and is attended with a mortality varying from 16·8 to 21·7 per cent., and the other an American type, which was prevalent during the same period in the United States, Canada and New South Wales and in the provinces in England, with a mortality varying from 0·45 to 5 per cent.

**Treatment.**—Owing to the risks attending the nursing of a smallpox patient in a private house, it is always advisable to have the case removed to a special hospital. When it is desirable that the patient should remain at home, a room should be chosen as far away as possible from the rest of the house. Except in very mild cases the diet should be restricted to fluids. Milk may be given plain or diluted, or rendered palatable by the addition of various proprietary preparations. Water should be taken freely. A day and a night nurse should



## SMALLPOX

be provided, who should be protected by recent vaccination, and members of the family and other contacts should also be vaccinated. The nurse should not be allowed to leave the house without changing her clothes, carefully disinfecting her face and hands, and washing her hair. The room should be well ventilated and stripped of all unnecessary fittings, and the temperature range between 60° and 64° F.

Careful attention must be paid in nursing to the toilet of the mouth and nostrils, especially in severe attacks. The mouth should be frequently cleansed with an antiseptic lotion, followed by painting with glycerin of borax. Prophylactic treatment of the eyes is important. To prevent the accumulation of purulent discharge the lids should be anointed with vaseline daily and the eyes washed out with boric-acid lotion. In severe attacks the hair should be cut very short or shaved.

No internal medicine has any influence in aborting the eruption, and drug treatment can only be symptomatic. The headache and pain in the back and limbs during the prodromal period may be treated by salicylates, aspirin or phenacetin, but in the excruciating back-ache which often attends the onset of hæmorrhagic smallpox nothing but an injection of morphia will give relief. The insomnia will require the administration of bromides and chloral, trional, or other hypnotics. To allay the vomiting which is often a troublesome symptom in the early stage, the milk should be peptonized or diluted with barley water or lime water, or given per rectum. The difficulty in swallowing caused by the eruption in the mouth and throat may be relieved by giving the patient ice to suck or by spraying the throat with a solution of cocaine.

No measures have proved of any avail in the treatment of hæmorrhagic smallpox, hæmostatics such as ergot, hazeline, or adrenalin being completely useless.

The cutaneous irritation may be relieved by warm alkaline baths, prepared by adding 2-10 oz. of soda to 30 gallons of water (Meara), or by cold compresses to which some vinegar has been added. A 2-per-cent. solution of carbolic acid is also said to have a very soothing effect (G. H. Mead). In children it will often be necessary to apply gloves to the hands and confine the arms in splints to prevent tearing open of the pocks. No local applications can be relied upon to abort or to prevent pitting, but the most successful results have been obtained by Welch and Schamberg from the use of tinc-

## SMELL, DISTURBANCE OF

ture of iodine, which they apply once or twice daily according to the sensitiveness of the skin, until the eleventh or twelfth day of the eruption, when it peels off in a thin parchment-like mask. An ointment dressing should then be substituted. This method appeared to make the pustules shrink, to hasten decrustation, and in some measure to lessen the pitting. The fætor may be checked by the application of a powder containing iodoform 2 parts, boric acid 10 parts, talc 28 parts, dusted on freely after a bath.

**Treatment of complications.**—On the onset of *laryngitis*, hot fomentations to the neck or a steam tent will be required, and if no relief is obtained by this means the question of intubation or tracheotomy will have to be considered. A 10-per-cent. solution of argyrol is useful in *conjunctivitis*. On the occurrence of *keratitis* and *corneal ulcer* an ointment containing hydr. ox. flav. 2 gr., atrop. sulph. 4 gr., and vaselin to 2 oz. should be applied twice daily.

**Red-light treatment** was recommended by Finsen, who claimed that the exclusion of all but red light from the wards in which smallpox cases were being treated had the effect of preventing suppuration of the vesicles and pitting, of abolishing the fever and of lowering the mortality. A trial of this method was made in the M.A.B. hospitals at Long Reach in 1903. The windows were covered with red material and the entrance to the experimental ward screened with thick curtains of Turkey twill. At night the only illumination was a red lamp. In no instance did the development of the eruption and the progress of the case differ from what might have been expected if the patients had been treated in the ordinary wards, and in some cases the gloom seemed to have a deleterious influence on the patients. Similarly unfavourable reports have been made by other observers.

J. D. ROLLESTON.

**SMELL, DISTURBANCE OF.**—The acuity of smell varies in different persons; in some it seems to be feeble, or even absent, from birth. Its loss, when acquired, may be due to local conditions in the nose, or to involvement of the nervous apparatus that subserves smell. The latter is rare, but occasionally anosmia results from a basal or cerebro-spinal meningitis, or is due to the compression of the olfactory bulb or tract by a tumour in the anterior fossa of the skull, or to a fracture in this region.

Any disease of the nasal mucous membrane may affect smell. Anosmia is often associated

## Snake-bite

with trigeminal paralysis, as this lessens the secretions and allows the nostril to become dry. It may be due to blocking of a nostril.

Perversions of smell are common in the neuroses and psychoses. Olfactory hallucinations are occasionally a very troublesome sequel of fracture of the anterior fossa of the skull and of traumatic injury of the olfactory tracts; these purely subjective sensations are almost invariably nasty and unpleasant. They cannot be relieved by treatment, but tend to disappear spontaneously.

GORDON HOLMES.

**SNAKE-BITE.**—Venomous snakes occur in most parts of the world, with the exception of New Zealand and the Oceanic Islands. They are divided by naturalists into two main classes, (1) the colubrines and (2) the viperines, which present differences in structure as well as in the toxic effects of their respective poisons.

In Europe small adders or vipers (*Vipera berus*, *V. aspis*, and *V. ammodytes*) are found, but are seldom dangerous to adult man, the fatalities recorded being mostly in children.

Asia is the home of the most poisonous colubrine snakes—for instance, two deadly species of viper—the “daboia,” *V. russelli*, and the “phoorsa,” *Echis carinatus*; the cobra, *Naja tripudians*, the king cobra, *N. bungarus*, and the kraits, *Bungarus caeruleus* and *B. fasciatus*. In Africa are found two species of cobra, *Naja haje* in the north and *N. flava* in the south, besides four species of viper—*Cerastes cornutus*, *Echis carinatus*, *Causus rhombatus*, and *Bitis arietans* or the “puff adder.”

The most poisonous snakes on the American continent belong to the Viperidæ and are representatives of the subfamily Crotalinæ. In North America are found various species of rattlesnake, *Crotalus terrificus*, *C. scutulatus*, *C. durissus*, and *C. horridus*, as well as the “copperhead,” *Ancistrodon contortrix*, and the “moccasin,” *A. piscivorus*. In South America there are various species of *Lachesis*, including the well-known “fer-de-lance,” *L. lanceolatus*.

Australia and Tasmania are rich in colubrines, among them the “death adder,” *Acanthopis antarcticus*, the “tiger snake,” *Notechis scutatus*, and also the black snake, *Hotechis pseudochis*.

The venoms secreted by the more harmful species vary greatly both in amount and toxicity. The venoms most potent in their effects upon man and experimental animals are those of the cobra, the Australian tiger

snake, and one of the sea snakes, *Enhydrina valakadien*.

The action of these lethal venoms depends upon certain ferments and lysins in solutions of modified proteins; all attempts to separate out the toxic principle in a pure state have so far failed. It is known, however, that the toxic bodies can withstand a considerable amount of heat and desiccation and, if dried at 20° C. in a desiccator over calcium chloride, can be kept indefinitely in a well-corked bottle.

The **symptoms** produced by snake-bite differ considerably in the two main groups. In colubrine poisoning signs of general systemic disturbance rapidly supervene, and the bitten part soon becomes angry and edematous. Nausea, vomiting with profuse salivation, and paralysis of the tongue and larynx follow. Death takes place when the respiratory centre becomes involved. In viperine poisoning extensive edema with blood-stained discharge and ecchymosis occurs at the seat of the bite. Constitutional disturbances pointing to partial paralysis of the cardiac mechanism supervene almost immediately. These phenomena may be transient; should they pass off, the tissue reaction at the site of the lesion becomes greatly aggravated, which results in suppuration and possibly gangrene of the affected part.

The **treatment** of snake-bite must be prompt and vigorous. It should be directed in the first place towards preventing, as far as is possible, the absorption of the poison, and secondly towards the neutralization of the generalized toxin. A ligature should therefore be tied around the limb immediately above the bite. If performed immediately, amputation of the affected part has proved to be effective.

As a means of destroying the toxins *in situ* the bite should be incised freely in the direction of the lymphatic and venous circulation, and crystals of permanganate of potassium vigorously rubbed in. Recently the injection of 20 c.c. of a 5-per-cent. solution of gold chloride in ox-bile has been advocated.

Mere suction of the wound is inefficacious. Alcohol and strychnine are probably useless as antidotes. General measures should be directed towards keeping the patient warm by means of hot-water bottles and blankets. If respiratory failure should supervene, artificial respiration must be resorted to.

The serum treatment of snake-bite is the

## SPA TREATMENT

most scientific as well as the most efficacious method. In practice, however, it has a very important drawback, namely, that while the serum may be specific for one particular species of snake, it may be impotent as regards other species. Attempts at the production of a multivalent serum acting against all snake venoms have met with no great degree of success. A practical method of overcoming the difficulty is to issue an antiserum effective against the most dangerous snakes in any given locality.

The injection should be made immediately, if possible within ten minutes, and by the intravenous route. A considerable amount is necessary to neutralize the toxins, and at least 100 c.c. should be given.

P. MANSON-BAHR.

**SODIUM CYANIDE POISONING** (*see* POISONS AND POISONING).

**SOFT SORE** (*see* ULCUS MOLLE).

**SOMNOLENCE** (*see* SLEEP, DISTURBANCES OF).

**SPA TREATMENT.**—A description of the various forms in which water is applied to the surface of the body for therapeutic purposes will be found in the article on HYDROTHERAPY. The present article is concerned with the classification of mineral waters, and with the affections in which their application, external or internal, or both, is efficacious.

Speaking broadly, the objects of a course of waters taken internally are—

1. To remove from the alimentary canal products of imperfect digestion, to stimulate the liver, to get rid of waste products by the bloodstream with subsequent elimination by the skin and kidneys—in short, to “wash out” the tissues.

2. To promote what was called by older writers the “alterative” effects of a course of waters. The main principle underlying all forms of treatment of chronic disorders is the promotion of a healthy reaction of the affected tissues—the stimulation of a reaction which presumably is inadequate. It is in the promotion of this “alterative” reaction that the spa treatment by means of waters and baths finds its application.

Baths may be grouped broadly into four classes, viz. (1) simple thermal baths, (2) baths depending mainly on the chemical constituents of the water, (3) gaseous thermal baths, (4) thermo-mechanical baths.

1. **Simple thermal baths.**—These are considered in the article on HYDROTHERAPY.

2. Baths depending mainly for their action on the **chemical constituents of the water** over and above their thermal effects—e.g.: brine baths, the brine acting as a chemical irritant, and a stimulant to the nerve-endings in the skin; mud baths, which are applied either locally or generally, and have the effect of stimulating the skin profoundly, and of increasing the body-heat; and sulphur baths, which cause cutaneous hyperæmia, cleanse the skin, have a germicidal action, and are powerful diaphoretics.

3. **Gaseous thermal baths.**—These owe their action to the stimulating effect of free CO<sub>2</sub> gas on the skin. They increase the peripheral circulation and promote diaphoresis.

4. **Thermo-mechanical baths.**—Such are needle baths, the Vichy and Aix douches, the Plombières treatment, and the “Tivoli” douche.

Before entering upon a classification of mineral waters, it may be pointed out that *altered climatic conditions* are of the greatest service as an auxiliary to treatment by such waters. In the past, perhaps too much attention has been paid to the height of the station above sea-level, without due recognition being given to the factors which profoundly modify the climate. Thus a spa at a moderate elevation only, with a free interchange of air, is often more bracing than another situated in a valley at a much higher altitude. Another factor is the *change of environment* for the patient, and a completely altered mode of life—early hours, modified diet, and regular exercise in the open air. Moreover, the physician secures more control over his patients, who are free from worry and business cares.

**Classification of mineral waters.**—It is customary to classify these waters according to their chemical constituents, and in the present state of knowledge no more scientific or precise method is possible. It will be noticed that waters are grouped according to their most important and most potent constituent—a principle, however, which is not free from difficulty, in that many waters contain more than one active substance, while of others the mineral content, indicated by chemical analysis, is negligible. Owing to limits of space, only the more important spas of each class will be mentioned, with special reference to those frequented by English and American patients. The classified list of waters is as follows:—

## SPA TREATMENT

1. Simple or indifferent thermal waters.
2. Mixed chloride waters.
3. Sulphur waters.
4. Alkaline waters.
5. Sulphated waters.
6. Chalybeate waters.
7. Calcareous waters.
8. Arsenical waters.

**1. Simple or indifferent thermal waters.**—The chief features of these waters are that they have a low mineralization, are very soft, and are naturally either warm or hot. They are more used for bathing than for drinking purposes.

*Bath* (84-750 ft.).—The Bath water emerges from the earth at a temperature of 114°-120° F. It is hypotonic and has a high degree of radio-activity. In addition to the usual balneo-therapeutic forms of treatment a prominent feature is the deep immersion bath, each bath containing 800-900 gallons of mineral water, in which the patient moves about freely. Bath is primarily an autumn and spring station, the climate being of a sedative character. The mean autumn temperature is 45° F., the spring 52° F.

*Buxton* (1,000 ft.). The water issues from nine springs at a temperature of 82° F., its most remarkable features being the large amount of nitrogen it holds in solution and its radio-activity. It is used largely for bathing purposes: (a) in large baths—at the natural temperature—in which the patient moves about freely, (b) in "hot" baths, in which with the natural waters is mixed a varying quantity of the same water artificially treated. Buxton is situated on limestone rock; the climate is dry and bracing.

*Matlock* (300-1,350 ft.).—The water, which is feebly mineralized, has a temperature of 68° F., and, in addition to the presence of a small amount of free CO<sub>2</sub>, contains a colloid substance which makes it well adapted for massage douching. The climate is mildly bracing. Matlock is notable among British spas in that the spa treatment is given in the hotels and hydros, and not in separate bathing establishments.

*Bagnères-de-Bigorre* (1,800 ft.) lies in a valley of the Pyrenees. In addition to the indifferent waters (temperature 88°-122° F.), the chief constituent of which is calcium sulphate, sulphur water is imported from a spring eight miles off. The baths are much used in the treatment of irritable conditions of the nervous system.

*Bagnoles-de-l'Orne* (750 ft.), situated in a hilly and well-wooded part of Normandy, has a thermal spring (La Grande Source, 80° F.) which is much used for bathing purposes, combined with massage, in the treatment of phlebitis and other affections of the peripheral circulation.

*Dax* (130 ft.), near Bayonne, well known as a winter health resort and bathing station, has radio-active waters (147° F.), used as full baths, douches, vapour baths, etc., also thermal mud baths which are partly vegetable and partly mineral in composition. The mud baths are given at high temperature in cases of stiffened joints, sciatica, fibrositis, and chronic rheumatism generally.

*Plombières* (1,300 ft.) is situated in a valley of the Vosges, surrounded by wooded slopes. The numerous springs vary in temperature from 55° to 165° F., and are radio-active. This spa is noted for its treatment of mucous-membranous colitis, typhilitis, appendicitis, chronic diarrhoea, and allied conditions, by means of lavage of the large intestine.

*Gastein*, Salzburg (3,130 ft.), is the possessor of numerous thermal, radio-active, feebly mineralized waters (80°-120° F.), and has a considerable reputation in the treatment of neurasthenia and disturbed and irritable affections of the nervous system generally. The waters are but little used for drinking purposes. The climate is of a sedative character, and this resort is very popular among elderly people who normally lead lives of great mental activity and strain.

*Schlungenbad* (900 ft.), in Nassau, and *Wildbad* (1,400 ft.), in the Würtemberg Black Forest, possess radio-active and feebly mineralized waters similar to those at Gastein, and are suitable for the same class of cases.

*Battaglia* (30 miles from Venice), in addition to its hyperthermal springs, is specially noted for its baths of fango, a volcanic mud, used either generally or locally in arthritis and gouty and rheumatic affections.

*Hammâm R'irha* (1,800 ft.), in Algeria, has hot springs of low mineralization (110° F.), which are utilized for bathing in large swimming baths. It possesses a fine winter climate.

**2. Mixed chloride waters.**—Common salt, being widely diffused through nature, is present in varying quantities in a very large number of mineral springs. As the blood-serum and body fluids contain NaCl, the function of tonicity is of special importance in this class of waters. They are termed "mixed chloride"

## SPA TREATMENT

as an indication that they contain chlorides other than that of sodium.

Many of the sulphur springs contain large quantities of the chlorides.

The *Droitwich* brine baths are practically a cold saturated solution of common salt (30 per cent.), and are utilized for bathing purposes only, in muscular rheumatism, sciatica, and allied affections. Brine swimming-baths are given, and, owing to the high specific gravity of the water, the patient can do graduated exercises which, on account of pain, he is unable to perform when not in the bath. This is a point of great importance in the treatment of sciatica and myalgias.

*Nantwich* has baths of the same character.

*Woodhall Spa* water contains, in addition to sodium chloride (20 parts per 1,000), iodides and bromides in small amounts. It is used for both drinking and bathing purposes. The spa has a special reputation in the treatment of uterine fibroids, chronic pelvic inflammatory exudations, vaginitis, and cervicitis.

*Bourbonne-les-Bains* (800 ft.), in the Vosges, has hot sodium-chloride springs (150°). The chief feature of the baths is the high-pressure douche of the mineral waters. There is a large military hospital at Bourbonne. The "cure" is indicated in cases of chronic rheumatism, chronic articular gout, sciatica, etc.

At *Chateaugayon* (1,300 ft.), Auvergne, are warm mineral springs containing the chlorides of magnesium and sodium. The waters are much used for stomach and intestinal lavage in the treatment of atonic dyspepsia, constipation, gastric catarrh, hepatic congestion, etc. Running-water baths are also administered, the water being rich in free carbonic-acid gas.

*Baden-Baden* (650 ft.), Black Forest, is renowned for its feebly mineralized sodium-chloride waters (124°–150° F.) containing traces of arsenic. The bathing establishments are very complete and luxurious, and all modern methods of hydrotherapeutic treatment are available.

*Homburg* (650 ft.) has many springs containing common salt in varying quantities with free carbonic-acid gas, and in some cases with chloride of calcium and bicarbonate of iron. The climate is dry and bracing, and the bathing establishments are of the first rank.

*Kissingen* (650 ft.), Bavaria, possesses cold CO<sub>2</sub> springs containing common salt and carbonate of iron. These are valuable in the treatment of anæmia, atonic dyspepsia, hepatic congestion, constipation, etc. Mud baths are

procurable, also "sool-spray baths," a vapour bath formed by the pulverization of the water.

At *Kreuznach* (340 ft.), near Bingen, are springs similar to those of Woodhall Spa.

*Nauheim* (400 ft.), Hesse, has five springs, two of which are used for drinking and three for bathing purposes. The most important of the latter is a warm common salt water containing CO<sub>2</sub>. The chief feature is the sprudel bath, used in the treatment of heart disease and circulatory disturbances; this, together with "resisted movements," forms the Nauheim treatment.

*Reichenhall*, in the Bavarian highlands, and *Bex* (1,400 ft.), in Switzerland, have brine baths of the same character, but not so strong, as those at Droitwich.

*Wiesbaden* (380 ft.), near Frankfort-on-the-Main, one of the most noted of German spas, is famous for its first-class bathing establishments, and clinics for the treatment of disease of the eyes, ears, etc. The chief feature of the springs is their high temperature. The Kochbrunnen (150° F.) is used for drinking and bathing purposes, inhalation, thermal and hyperthermal baths.

*Salso Maggiore* (500 ft.), in the province of Parma, Italy, has a cold brine which contains compounds of iodine, brine, and petroleum, the last-named constituent rendering it unsuitable for drinking. It is used for both bathing and inhalation. Mud baths are employed, particularly in the form of fango poultices.

**3. Sulphur waters.**-- A large number of springs are grouped in this class because, although differing widely in many ways, they all contain sulphur in the form either of sulphuretted hydrogen or of sulphides. They are scattered all over the world, some (and chiefly in volcanic districts) being thermal (e.g. in the Pyrenees and New Zealand), others cold (e.g. in Great Britain). They have been used for drinking and bathing purposes for centuries, and have a great reputation in the treatment of chronic skin affections, gout, rheumatism, and various forms of liver and stomach disorder. Sulphuretted hydrogen quickly finds its way into the tissues from the stomach, being eliminated largely through the skin, kidneys, and bronchial mucous membranes.

*Harrogate* (600 ft.) possesses a great variety of mineral springs, including a number containing sulphur combined with varying proportions of salt. Thus the Old Sulphur Well contains 0.7 vols. per mille of sodium sulphide and 13 parts of sodium chloride with 37 vols.

## SPA TREATMENT

of sulphuretted hydrogen per litre. Other springs contain much less salt. It follows, therefore, that some of the springs are hypertonic with reference to the blood-serum, while others have an astringent effect upon the intestinal mucous membrane. There are also several saline springs in which iron is present either as the chloride, carbonate, or sulphate. Although the elevation is moderate the climate is particularly bracing, owing to the open position of the town on a moorland plateau with free interchange of air. The balneological equipment is very complete, including mud, sulphur, douche, vapour, and other baths, intestinal lavage, etc. The "cure" is indicated in cases of gout and other disorders of metabolism, functional liver derangement, cholelithiasis, eczema, psoriasis, and other chronic skin diseases, sciatica, fibrositis, arthritis, high blood-pressure, etc.

*Llandrindod Wells*, in Mid-Wales, has (1) sulphur and saline, (2) common salt, and (3) chalybeate springs. The waters of the first two groups, which are of low mineralization, are hypotonic with reference to the fluids of the body. The chalybeate spring contains the carbonate of iron. The altitude at the highest part is 700 ft., and the climate is bracing.

*Strathpeffer* (150-300 ft.), in the Scottish Highlands, has springs of which the leading characteristics are the large percentage (69 vols. per mille) of sulphur present in the form of sulphuretted hydrogen, and the low percentage of sodium chloride. The climate is mild owing to the position of the spa in a valley sheltered by mountains.

*Aix-les-Bains* (860 ft.), Savoy, one of the most popular spas in Europe, has warm sulphur springs similar in character to those at Strathpeffer. The water is chiefly used for bathing purposes, and the characteristic feature of this spa is the Aix douche, a combination of massage and douching (see HYDROTHERAPY). Chronic affections of the nose and pharynx are treated with the natural vapour of the hot sulphur springs at Marlioz, close to Aix. The climate is mild and warm.

*Barèges* (4,000 ft.), in the Pyrenees, possesses a number of warm sulphur springs. The waters are unctuous owing to the presence of organic matter (to which the name *barégine* has been applied), and are consequently particularly suitable for wet massage. Barèges possesses a state military hospital, the baths having a great reputation in the treatment of injuries received on the battlefield.

*Luchon* (2,000 ft.) and *Cauterets* (3,200 ft.), both in the Pyrenees, have warm sulphur springs. The chief feature at both spas is the treatment of chronic naso-pharyngeal catarrh by inhalations, pulverization, gargles, etc.

*Aix-la-Chapelle*, or Aachen (530 ft.), is noted for its hot sulphur springs, which contain sulphuretted hydrogen, sodium sulphide, and common salt. It has a worldwide reputation in the treatment of syphilis by means of mercurial inunction following sulphur baths.

*Schinzach* (1,100 ft.), in the valley of the Aar, has sulphur springs containing sulphuretted hydrogen at a temperature of 82°-95° F. The baths are much used in psoriasis and other scaly skin affections in which a macerating effect is desired.

*Acqui*, in Northern Italy, possesses hot saline springs, but its most important feature is its fango baths, volcanic mud being mixed with mineral water.

At *Helouan-les-Bains*, in Egypt, 16 miles south of Cairo, are sulphuretted saline sulphur waters similar in character to the Old Sulphur Well, Harrogate. The climate is dry, warm and sunny, and the air remarkably free from pollution owing to the situation of Helouan in the desert. It is an important winter resort for patients suffering from arthritis and chronic rheumatism.

4. **Alkaline waters.**—All the waters of this group owe their alkalinity to the presence of sodium bicarbonate, and are largely used for internal administration in the treatment of conditions of the stomach and bile-passages, and when an antacid flushing of the mucosa is desired.

*Vichy* (850 ft.) has many cold and thermal isotonic alkaline waters containing CO<sub>2</sub>, and traces of iron and arsenic. L'Hôpital (81.5° F.) is used in gastric maladies for drinking and lavage; Celestin, a cold spring, diuretic in action, for gout and renal disturbances; La Grande Grille (106° F.), for hepatic affections. The Vichy douche is used extensively. At Vichy there is a military hospital for officers and men invalided with malarial affections after service in the French colonies.

*Royat-les-Bains* (1,480 ft.), in the Auvergne, has an alkaline water which contains an appreciable amount of sodium chloride and free CO<sub>2</sub>. It is used internally, for inhalation, and as effervescing baths in gouty affections involving the skin and in bronchial catarrh.

*Ems* (260 ft.), near Coblenz, with springs of the same character as those at Royat, has a

## SPA TREATMENT

great reputation for the treatment of chronic catarrh.

*Carlsbad* (1,200 ft.) and *Marienbad* (1,900 ft.) are the two most important representatives of the class of alkaline waters which, containing sodium sulphate in addition to sodium bicarbonate, have a purgative action by increasing intestinal peristalsis. The Carlsbad waters are hyperthermal waters, gaseous and radio-active. The "cure" is indicated in cases of abdominal plethora, over-feeding, obesity, constipation, liver affections, and certain types of glycosuria. The waters of *Marienbad* are of the same character, except that they are cold and, containing more sodium sulphate and  $\text{CO}_2$ , are more aperient. An important feature of this spa is the mud bath. The "cure" is suitable for the same types of cases as Carlsbad, and has a particular reputation in the treatment of obesity.

5. **Sulphated waters.**—Mineral waters of this class are distinguished by the amount of magnesium sulphate and sodium sulphate they contain; they are sometimes called bitter waters because of the taste of the magnesium sulphate. The more active members of the group are not drunk at their source, but bottled; such are *Apenta*, *Rubinat*, *Carabana* (Spain), and *Friedrichshall*. They are of service in cases of sluggish portal circulation, hæmorrhoids, lithiasis, etc.

*Cheltenham* (150 ft.) has springs containing magnesium sulphate and sodium chloride, with a consequent mild aperient effect; also chalybeate waters.

6. **Chalybeate waters.**—Iron, being widely diffused through nature, is contained in the majority of mineral waters, but in this class are included only those springs in which iron is present in appreciable amount. A course of chalybeate waters is indicated in cases of debility associated with chlorosis. Those containing carbonic-acid gas are utilized for bathing purposes, but, inasmuch as there is no cutaneous absorption of iron, their action is no doubt due to the effect of the gas upon the skin.

The springs at *Harrogate* contain iron in the form of ferrous carbonate, and the chloride in varying amounts, associated with sodium and other chlorides.

*Flitwick*, in Bedfordshire, has a cold spring containing the persulphate of iron.

*Spa* (1,000 ft.), Belgium, is noted for its cold springs containing the bicarbonate of iron and much free carbonic-oxygen gas. They are used

for both bathing and drinking purposes. *Spa* has a reputation in the treatment of menstrual irregularities, vaginal and uterine catarrh, chlorosis, etc.

*Schwalbach* (950 ft.), Germany, possesses non-thermal bicarbonate-of-iron waters, which are used for bathing purposes and for vaginal douching.

7. **Calcareous waters.**—These springs contain the carbonate or sulphate of calcium and carbonate of magnesium, with or without free carbonic-oxygen gas, as their chief ingredients; some contain sodium bicarbonate in addition. Speaking generally, they are strongly diuretic in action, and by increasing renal elimination reduce high arterial tension. Prescribed in large doses, their beneficial effect is chiefly due to the washing-out of the tissues. They are used largely in cases of chronic bronchial catarrh and catarrh of the bladder and urinary passages, and have a very definite action in lessening secretion of the mucous membranes involved.

*Bath.* (See under Class 1, p. 223.)

The waters of *Contrexéville* (1,150 ft.), in the Vosges, are of the cold calcareous type, and are markedly diuretic in action. This spa has a great reputation in the treatment of renal calculus, gravel, chronic cystitis, oxaluria, etc.

*Vittel* (1,100 ft.), also in the Vosges, has waters of the same character as those of *Contrexéville*.

*Wildungen* (980 ft.), the German *Contrexéville*, has non-thermal gaseous waters which contain the bicarbonates of sodium and magnesium, and are utilized in the treatment of prostatic enlargement, urinary lithiasis, and vesical catarrh.

8. **Arsenical waters.**—In this group are included a small number of mineral springs which contain an appreciable quantity of arsenic; their physiological and therapeutic action is the same as when arsenic is administered in pharmaceutical preparations.

*Ronccigno* and *Levico*, in the Tyrol, are the strongest arsenical springs in the world, the former containing 42.6 mg. of arsenic trioxide per litre.

*La Bourboule*, in the Auvergne (2,770 ft.), has a spring (temperature 136° F.) containing sodium bicarbonate, sodium chloride, and 0.028 grm. of sodium arsenate to the litre. The "cure" is recommended in psoriasis, eczema, ichthyosis, acne, and many other skin affections, in scrofulous conditions, malarial

## SPASM, CAUSES OF

cachexia, asthma in children, and certain types of glycosuria.

*Mont Dore* (3,400 ft.), the highest spa in France, has hyperthermal feebly-mineralized alkaline waters containing a trace of arsenic. A chief feature of this spa is its *salles d'aspiration*—chambers filled with the compressed vapour of the water, and used in the treatment of asthma and catarrhal conditions of the respiratory passages.

W. BERTRAM WATSON.

**SPASM, CAUSES OF.**—A spasm is definable as a tonic contraction of a muscle or group of muscles, caused by an increase of excitability of the muscles themselves, by stimulation of their motor nerves, or as a reflex evoked by an appropriate sensory stimulus. The term has, however, been very loosely applied to designate tonic muscular contractions of the most diverse origin; even the various co-ordinated movements exhibited in tics have been called spasms. These spasmodic contractions unassociated with an organic lesion and under volitional control are considered in the articles on HYSTERIA AND HYSTERO-EPILEPSY and TICS AND HABIT SPASMS.

**Spasm of muscular origin.**—The prolonged ischaemia of a muscle produced by tight bandaging will cause a spasmodic contraction of purely muscular origin, due probably to changes in the muscle induced by the heaping-up of products of muscular metabolism. Similar myogenic contracture follows the long-maintained tonic contraction of local tetanus, hysterical contractures, and the unopposed contracture due to paralysis of an antagonist muscle. These myogenic spasms are independent of the central nervous system and may not be resolved by section of the motor nerves. They pass slowly away when the lymph- and blood-circulation are restored by passive movements and massage.

**Spasm due to hyperexcitability of a motor nerve.**—Tetany (q.v.) is characterized by spasmodic contractions due to hyperexcitability of the peripheral nerves. Facial spasm due apparently to such hyperexcitability is a comparatively rare disorder; it begins with irregular fibrillary contractions of the facial muscles on one side, which increase in frequency and intensity till finally the whole facial musculature on one side is contracted spasmodically. The spasm may last for weeks or months, or occur in bouts of short duration. Occasionally hearing is affected also. The absence of other nervous symptoms, the involvement of both

upper and lower facial muscles, and the fibrillary tremor accompanying the spasm serve to distinguish it from facial spasm due to a cortical discharge, or the facial spasm of hysteria.

Wounds causing **cicatricial irritation of a motor nerve** occasionally give rise to spasm.

**Hyperexcitability of the motor nerve-cells** in the spinal cord is the cause of the spasm of tetanus.

Spasm due to **irritation of an afferent nerve**, and therefore reflex in nature, is, as a rule, accompanied by pains and is discontinuous. The contractures frequently observed after wounds of the extremities in the late War were originally considered to be reflex in nature, but have been proved to be really due to muscular change following on the maintenance of an hysterical contracture. The muscle is poisoned by the retention of waste products of metabolism due to circulatory disturbance associated with the maintenance of a long-sustained contracture.

Reflex spasm evoked by **hypersensibility of the afferent nerves** occurs in poisoning by strychnine.

**Lesions of the spinal cord** cutting off the cerebral inhibitory impulses may permit an excessive response of the motor neurones to sensory stimuli which is spasmodic in nature.

**Irritation of the afferent nerve-trunks** in their passage through the meninges may cause spasmodic contractions in meningitis, the spasm being often most emphatic in the retractor muscles of the head and spine.

Spasmodic contractures of the **unstriated muscles of the viscera** may take place as a response to direct excitation, but it may also be a reflex response of the vegetative nervous system to irritation in another portion of the viscus.

Hyperexcitability of **specific visceral systems** may exist, as in vaginismus and spasm of the pylorus.

**Spasms of cerebral origin** may be due to irritative lesions of the cerebral cortex. In such cases there are usually symptoms pointing to involvement of the pyramidal tracts. The distribution of such spasms corresponds with the area of the musculature governed by the cortical zone affected, but the spasms occasionally spread till gradually the whole skeletal musculature of one side of the body is involved in what is known as a Jacksonian fit. Spasmodic contractions of the hand muscles may occur in **occupation neuroses** such as writer's cramp.

F. L. GOLLA.



## SPASMOPHILIA

**SPASMODIO DYSMENORRHEA** (*see* DYSMENORRHEA).

**SPASMODIC TIC** (*see* TICS AND HABIT SPASMS).

**SPASMODIO TORTICOLLIS** (*see* TORTICOLLIS AND WRY-NECK).

**SPASMOPHILIA.**—A term, little used in Great Britain, which has been applied by American physicians to a constitutional state or diathesis the chief manifestations of which are convulsions, tetany, facial irritability and laryngismus stridulus. It is met with almost entirely in infants and young children. Three factors co-operate in its causation: (1) An underlying nervous instability, often inherited. (2) A general constitutional cause which is at present undetermined but in which defective functioning of the parathyroids possibly plays a part. Rickets is not uncommonly present. Reduction in the calcium-content of the blood seems to be a constant feature, and explains the undue excitability of the nervous system. (3) An exciting cause such as dyspepsia, or acute infections or intoxications. Offensive and otherwise abnormal motions often immediately precede such prominent manifestations as tetany and laryngismus. The spasmophilic child is petulant, excitable, restless, and irritable. (*See also* TETANY; and LARYNGISMUS STRIDULUS.)

FREDERICK LANGMEAD.

**SPASMUS NUTANS** (Nodding Spasm).—Nodding movements of the head which occur in early infancy, accompanied by nystagmus, and probably due to defective co-ordination.

**Etiology.**—Spasmus nutans is usually seen first in infants between the ages of four and twelve months, and generally disappears before the third year. The association of nystagmus with the nodding implies imperfect co-ordination between the movements of the ocular and cranio-cervical muscles. Guthrie held that Deiter's nucleus and its connexions with the oculo-motor nuclei beneath the corpora quadrigemina, and with the anterior column of the cord, through the vestibulo-spinal tracts, are probably the seat of the disturbance. The frequent incidence of spasmus nutans during December and January, and its occurrence in children who live in dark houses, led to the belief that the nystagmus is due to the darkness, or to the child's attempt to fix a bright object in a dark room, and that the

## SPASMUS NUTANS

nodding is secondary. Dark rooms and dark months, however, are not always associated with it. Many of the infants are rickety, but others are not. Dentition and gastro-intestinal disturbances are other suggested causes, but the association is not clear. Nervousness and supersensitiveness to noise have been noted, and in this way the nystagmus may be related to that caused by syringing the ear, sound itself being sufficient to produce the phenomenon.

**Symptoms.**—The head movements may be up and down or from side to side, but are usually oblique. The nystagmus may be horizontal, vertical, rotatory, oblique, or even convergent. The nodding and nystagmus do not always correspond, nor are they always synchronous, for one may precede or succeed the other. Though usually bilateral, the nystagmus may be confined to one eye. Sometimes the nodding may interfere considerably with nursing, the teat being grasped with difficulty and often dropped. When looking at objects the child is prone to tilt its head to one side, as though hemianopic.

**Diagnosis.**—Spasmus nutans must be distinguished from hereditary *nystagmus*, with which head-nodding may occur. Both appear at about the same time, but the hereditary form of nystagmus persists throughout life. It affects both sexes, but is usually transmitted through the female to the male. The subjects are very fair, with pale discs and blue irides, and were regarded by Nettleship as mild albinos. Almost always hypertrophic astigmatism is present. It may be impossible to distinguish the nystagmus when nodding movements are absent from that which accompanies grave defects of vision, except by ophthalmoscopic examination. "Head-rolling" is a different condition. The head is pressed against the pillow and rolled deliberately from side to side until perhaps a bald patch results. Its common causes are intestinal disturbances, otalgia and rickets.

**Treatment.**—Attention to hygiene and the diet are generally all that is necessary. If the motions are abnormal a mixture containing 5-10 min. doses of castor oil may be employed. Potassium bromide in small quantities often leads to cessation of the nodding, which is itself of little consequence.

FREDERICK LANGMEAD.

**SPASTIC PARALYSIS OF INFANTS** (*see* CEREBRAL DIPLEGIA).

## SPEECH, DISTURBANCES OF

**SPASTIC PARAPLEGIA** (see PARAPLEGIA, SPASTIC).

**SPEECH, DISTURBANCES OF.**—Under this heading are considered Dysarthria and Anarthria, Aphonia, and Stammering.

### DYSARTHRIA AND ANARTHRIA

When there is a lesion in any part of the association apparatus between the various cortical centres concerned with the faculty of speech, the resulting defect is one of aphasia (q.v.). When there is a lesion in the projection path or paths from the motor speech-centre in Broca's area of the frontal lobe to the secondary centres for the movements of speaking—the nuclei of the seventh, tenth, and twelfth cranial nerves, situated in the pons and medulla, which supply the muscles of tongue, palate, lips, etc.—the clinical condition is one of dysarthria or anarthria.

These terms must be restricted to defects of articulation and phonation, of outward speech, while aphasia is a defect of inward speech.

The projection paths just referred to, cortico-pontine and cortico-bulbar motor tracts, originate in the corresponding motor centres, which are situated in the lower part of the precentral gyrus in each hemisphere, and in the adjacent operculum Rolandi, i.e. the part of the precentral gyrus which overhangs the anterior portion of the island of Reil. The fibres are collected together into a special bundle, the geniculate bundle, which skirts the lenticular nucleus and passes through the knee of the internal capsule, whence it descends to the crus cerebri, occupying the inner fifth of that structure; the fibres then gradually separate into smaller bundles and, crossing the midline at varying levels, reach the contralateral motor nuclei above specified.

Dysarthria and anarthria are caused by bilateral lesions anywhere on this path, but occasionally also by unilateral lesions—which can be understood, as there is probably a double representation in the cortex of each half of the body. The common diseases associated with dysarthria are bulbar palsy, pseudo-bulbar paralysis, myasthenia gravis, poliomyelitis inferior, and vascular lesions of the basal ganglia, crus, pons, or medulla. Dysarthria may also ensue if there is defect in the co-ordination of the mechanisms concerned, without actual palsy. Ataxia of articulation may occur in cerebellar disease;

and in Friedreich's disease, disseminated sclerosis, ponto-cerebellar tumours, etc., dysarthria from impairment of controlling mechanisms may arise.

The **prognosis** and **treatment** of dysarthria are obviously those of the underlying pathological condition, and the reader is therefore referred to the articles dealing with those subjects.

### APHONIA

The term aphonia is used to signify a condition of whispered speech. Its most common occurrence is as a symptom of hysteria, monosymptomatically or otherwise. The treatment of the hysterical variety is that of the psychoneurosis itself.

Aphonia, however, in mild or severe degree, may occur from local conditions impairing the function of the recurrent laryngeal nerves; dysphonia or aphonia, also, may be an accompaniment of dysarthria.

### STAMMERING

Stammering, or stuttering, is a neurosis of articulation; it is an impairment of the function of outward speech, of a peculiar nature, but central in origin and psychomotor in type. It occurs in predisposed individuals, who not infrequently show other signs of psychical instability.

The usual cause of stammering is a shock or fright, a severe fall, an exciting or alarming experience; it is often associated with the existence of a mental state of general anxiety. Undoubtedly imitation, intentional or unintentional, is an important etiological factor. Recent claims by the Freudian school would make a sexual factor paramount in the development of stammering.

**Symptomatology.**—From this point of view the condition reveals itself by cramps or spasms of the muscles of phonation and articulation, while additional voluntary or involuntary muscular contractions in neighbouring or distant groups are almost habitual. The patient may stick over certain letters only, or certain words. He is always afraid that he will not be able to articulate correctly, with the result that he defeats his own efforts to speak smoothly. This element of lack of confidence is sometimes strikingly apparent. Stammerers often are able to read aloud with someone in complete ease, but the moment the accompanying voice ceases and they are left to their own devices they stutter. The timidity which so many stammerers show

## SPEECH, DISTURBANCES OF

exhibits itself in diverse ways—bashfulness, strained feelings towards others in society, blushing, etc. Scripture distinguishes three stages in stammering: (1) the habit stage, in which the child is merely copying and the stammering is dropped as soon as the cause is removed; (2) the fright stage, in which “the fear of displeasing and of appearing ridiculous” is uppermost, and absolute speechlessness may result; (3) the stage of indifference, in older patients, who stammer because the habit is firmly fixed, and not because they are embarrassed.

The **treatment** of stammering must be directed to the two sides of the neurosis—i.e. to the actual articulatory defects and to the psychopathic state behind it.

It has often been remarked that stammerers can sing what they cannot say in an ordinary speaking voice. The explanation is that singing does not awaken the emotional complex that is associated with the speaking voice; and accordingly various methods have been devised to lead the patient to speak in an unusual voice, low-pitched or high-pitched, as the case may be. They are objectionable, as Scripture says, because they leave the patient with a queer voice which is sometimes worse than the disease.

A great number of exercises for breathing and articulating, which may be found in any of the modern textbooks dealing with stammering, have been devised to aid in the re-education of the voice. More importance, however, must undoubtedly be placed on the treatment of the mental factor in the condition. To this end methods must be utilized of a more general nature, psychotherapeutic methods which need not be further particularized in this place. The method of psycho-analysis will prove of service in suitable cases, and good results may follow, but its application to all and sundry cases of stammering in children, as has been recently advocated by one of its enthusiasts, is to be deprecated, especially since excellent results have often been obtained by other procedures.

S. A. KINNIER WILSON.

**SPERMATORRHEA** (*see* SEXUAL FUNCTIONS, MALE, DISTURBANCES OF).

**SPHENOIDAL SINUS, DISEASES OF** (*see* SINUSES, ACCESSORY AIR, DISEASES OF).

**SPHYGMOMANOMETER** (*see* BLOOD-PRESSURE).

## SPINA BIFIDA

**SPINA BIFIDA.**—A condition of imperfect development of the spinal canal, usually accompanied by protrusion of some of the spinal contents.

**Etiology.**—Very little is known as to causation. It is a condition of arrested development, often accompanied by mal-developments in other parts of the body. It is found almost equally in males and females, and occurs about once in a thousand births.

**Pathology.**—The spinal cord is formed by the raising up and meeting of the medullary folds of embryonic epiblast. Mesoderm grows round the neural tube and constitutes the basis of the future meninges and vertebral column. The neural tube closes first in the dorsal region, next in the cervical portion, and lastly in the lumbo-sacral portion of the spinal canal.

When the neural tube does not unite but the medullary substance is exposed as a flat vascular area the condition is known as *rachischisis*, which may involve the whole or part of the cord.

Of spina bifida proper there are four pathological varieties:

1. *Meningocele*, or protrusion of the membranes of the cord through a gap in the vertebral column. Fluid collects within the protruded portion of the membrane.

2. *Syringo-myelocoele*, or dilatation of a portion of the spinal cord, which projects through the vertebral gap.

3. *Meningo-myelocoele*, the projection of a portion of the pia mater and spinal cord, which is spread over the summit of the swelling and under which fluid collects.

4. *Spina bifida occulta*, or defect of the neural arches unaccompanied by any protrusion of the vertebral contents.

Very rarely the defect of the bone affects the bodies of the vertebræ and a spina bifida anterior results. Hare-lip, talipes, and hydrocephalus are among the bodily defects that often accompany spina bifida.

**Symptoms.**—The symptoms present in a case of spina bifida are—

- (a) The presence of a swelling over the spine, noticed at birth. In the occult variety no protrusion may exist, but there is often a small tumour, lipoma or angioma, over the site of the defect, or there may be a tuft of hair.

- (b) Nerve symptoms, due to involvement of the end of the cord and nerve-roots, e.g. paralysis or spastic condition of lower extremities, incontinence of urine and feces.

- (c) Associated deformities such as hare-lip or

## SPINAL CARIES

**hydrocephalus.** *Spina bifida occulta* may give rise to no symptoms till puberty, when cord symptoms may develop.

**Diagnosis.**—Except in the occult and anterior varieties, no difficulty exists in diagnosing *spina bifida*. Differentiation between the varieties is usually possible. In *meningo-myelocele* there are three zones observable—in the centre the red nervous matter, outside this the thinned-out pial covering, at the periphery the thinned skin merging gradually into normal skin. In *meningocele* the vertebral gap is usually small and the swelling may be pedunculated. In *syringo-myelocele* the presence of nerve-elements in the sac may be seen by transillumination, and pressure in the sac may cause bulging of the fontanelle. Nerve symptoms often accompany this variety.

The X-rays will reveal a *spina bifida* anterior or occulta.

**Prognosis.**—In *spina bifida occulta* the prognosis is good, in *meningocele* promising, in *syringo-myelocele* hopeless.

**Treatment. Palliative.**—Protect the part and prevent ulceration as far as possible. Aspirate aseptically if there is danger of the thinned cover bursting.

**Curative.**—Occasionally benefit has accrued from injecting the sac with  $\frac{1}{2}$ –1 dr. of Morton's fluid (iodine 10 gr., potassium iodide 30 gr., glycerin to the ounce).

Operation is advisable in cases of *meningocele*. It should not be undertaken until the child is a few years old. The sac may be removed, the opening in the membranes securely sewn up, and if necessary a musculo-fascial flap fashioned to make good the bony defect.

It is doubtful whether much good results from operating on either *syringo-myelocele* or *meningo-myelocele*. Absolute contraindications to operation are *hydrocephalus*, *paraplegia*, and other grave associated deformities.

ZACHARY COPE.

**SPINAL CARIES.**—The name spinal caries is reserved for tuberculous disease of the vertebræ. Inflammation of these bones is in extremely rare instances due to *B. typhosus*, the gonococcus, or the staphylococcus.

**Etiology.**—As in tuberculosis of other regions, general debility with anæmia is an important predisposing cause, and as a rule there is no history of injury. The disease is more common in children than in adults, and begins especially between the ages of 5 and 15, but cervical caries is often seen in very

young children. The actual cause, the tubercle bacillus, gains access to the blood-stream by way of the respiratory or alimentary tract, and especially the tonsil. While in most cases the only obvious focus is in the spine, associated disease of the lungs, joints, or genito-urinary tract is not very uncommon.

**Pathology.**—In the neck the inflammation usually begins as an arthritis, and spreads later to the bodies of the vertebræ; in the dorsal and lumbar regions it begins either in the periosteum covering the front of the vertebral bodies, or in the anterior part of the cancellous tissue close to the upper or lower surface. In children the focus starts in the anterior part of the diaphysis in relation to the upper or lower epiphysis.

As the inflammation spreads it takes one of two courses. In the commoner type of case it extends back into the body of the affected bone, which becomes carious and eroded; the adjacent intervertebral disc is then destroyed, and the disease spreads to the body of the next vertebra; the two carious bones then come together and collapse under the weight of the head and trunk and the pull of muscles, and an acute angulation of the spinal column is produced; gradually other vertebræ above and below are involved. Meanwhile the lateral processes are pressed together and become ankylosed, causing permanent fixation of the diseased region; by the immobility thus produced a chance is afforded for arrest of the disease.

In the less common variety the disease begins as a periostitis, and spreads under the anterior common ligament so as to involve several vertebræ; a gradual curve of the spine is produced. This type is commoner in adults, in whom the cancellous bone offers a greater resistance than in children.

Progress of the inflammation results in caseation, and cold abscesses form and travel in various directions, guided by the muscles and fasciæ. If an abscess reaches the surface and invades the skin, a sinus forms and becomes invaded by secondary septic bacteria; these cause an acceleration of the disease, toxæmia is increased, and eventually lardaceous disease may supervene.

In certain chronic cases in adults the disease takes the form of a *caries sicca* and remains limited to the bodies of one or two vertebræ, producing few symptoms and little deformity; this type occurs particularly in the lumbar region.

Spread of inflammation into the spinal canal

## SPINAL CARIES

may produce paraplegia, either from oedema of the cord, from constricting fibrosis, or in rare instances from the pressure of an abscess.

**Symptomatology.**—The first thing noticed may be general debility with anæmia and wasting. Pain is soon complained of; it is of an aching character at the site of the disease, worse in the evening and after exertion, and relieved by lying down; or it may be referred along the peripheral nerves, and take the form of earache or pain in the occipital region, chest, shoulders, or arms in cervical caries. In thoracic caries there may be "stomach ache" or pain in the chest; and in lumbar caries there may be sciatica or pain down the front of the thighs. Starting-pains at night occasionally occur. On examination, pain in the region diseased is produced by jarring the spine, by pressure over the transverse processes of the diseased bones, and in later stages by pressure applied over their spinous processes.

Rigidity is present from the earliest stage of the disease; there is a general rigidity of the erector spinæ group of muscles—which disappears to some extent after a few days' rest in bed—in addition to the complete absence of movement between the vertebræ actually diseased. This gives the most important physical sign in early cases; in adults and older children it is tested for by making the patient, sitting or standing, alternately flex and extend his spine, while the doctor with his fingers on the spinous processes tests their mobility systematically the whole way down the vertebral column. Young children should be examined lying prone; the doctor, holding the legs in one hand, bends the spine from side to side, while with the fingers of his other hand he examines the mobility of all the segments of the spine in turn. Localized rigidity can be easily overlooked, so it is not sufficient to get a general view of the flexibility of the spine as a whole. In cervical caries, which is quite common in young children, spasmodic torticollis is often the earliest symptom.

Deformity takes the form of kyphosis, with a secondary compensating curve in some cases. The commonest curve is an angular one, the spinous process of the highest vertebra diseased making a marked prominence; but in the diffuse form of the disease met with in adults there is a gradual kyphotic curvature involving several vertebræ. Occasionally in the thoracic region the deformity presents itself as a lessening of the normal curve, giving a rigid flattening, the

so-called "poker-back"; in lumbar caries also there may be a flattening, from loss of the normal concavity. In disease of the cervico-dorsal and upper dorsal regions there is sometimes very great deformity, so that the ribs are crowded together and the sternum projects forwards, while the viscera of the abdomen and thorax are displaced, in some cases causing dyspnoea. In all the forms and situations of the disease the spinous processes of the affected bones are felt to be abnormally close together. Lumbar and cervical lordosis occurs as compensatory curvature in thoracic caries, and scoliosis is occasionally present, especially in cervical caries. In disease of the axis vertebra, fracture of its odontoid process may occur, leading to sudden death.

Abscesses are present in the later stages, and give rise to fluctuant swellings—at first deep, later superficial—appearing in various situations according to which part of the spinal column is diseased. In cervical caries they appear as retro-pharyngeal abscess and in the posterior triangle or axilla, in the last two situations usually being masked by enlarged lymphatic glands. In disease of the thoracic vertebræ there may be intercostal abscess tracking along an intercostal nerve to point laterally, dorsal abscess presenting posteriorly at the side of the erector spinæ group of muscles, or mediastinal abscess. In dorso-lumbar and lumbar caries, iliac abscess appearing above Poupart's ligament, psoas abscess below Poupart's ligament, lumbar abscess at Petit's triangle, and ischio-rectal abscess are seen, while the infection may spread to the sacro-iliac joint or the hip.

Paraplegia occurs especially in cervico-dorsal and high dorsal caries; it is first evident by an exaggeration of the reflexes—increased knee-jerks, ankle-clonus, and an extensor plantar response; later there may be adductor spasm, or even scissor-legged deformity and spastic talipes equinus. In lumbar caries a flaccid paralysis may supervene, the lower limbs becoming lax and powerless. There may be retention of urine with overflow, and occasionally incontinence of urine and fæces; cystitis is apt to occur when there is retention. Anæsthesia is very rarely present.

**Diagnosis.**—The sign upon which most reliance must be placed is rigidity, and by its means spinal caries is to be distinguished from *adolescent scoliosis* and *kyphosis*, and from other forms of curvature. *Malignant disease of the*

## SPINAL CARIES

*vertebræ* is rare; it is characterized by very severe pain which is not relieved by rest, and there are likely to be areas of anæsthesia. *Osteo-arthritis of the lumbar spine* is usually preceded by injury; it occurs in older people, and it causes unilateral referred pains in most cases. The cold abscesses of spinal caries simulate many things, especially *femoral hernia* and *lipoma*, but if the spine is examined carefully in doubtful cases, mistakes will not occur.

The most valuable method of settling the diagnosis and the extent of the bone disease is by X-rays, and by this means also mediastinal abscess can be detected; in early cases, particularly in the thoracic region, a lateral negative is necessary to show a limited bone focus.

**Prognosis.**—Under modern methods of treatment the prognosis is good, unless the disease has progressed to sinus-formation or has spread to other regions, especially the hip-joint. When there are several sinuses, profuse suppuration goes on and the patient becomes wasted and cachectic, with grave risk of bedsores; in such cases lardaceous disease is likely to supervene.

In cervical caries, especially in young children, the outlook is not good; the disease may progress rapidly, and sudden death is not uncommon.

In every case of spinal caries cure will only result from prolonged careful treatment, which must be continued for at least a year after every sign and symptom of active disease has disappeared; in adults this period must be even longer. If the disease is arrested before puberty, recrudescence is unusual. In all cases of marked angulation the deformity is likely progressively to increase even after the tuberculosis is cured, unless a permanent support is used.

**Treatment.**—The general treatment follows the lines laid down for all forms of tuberculosis—fresh air, mental and physical rest, tonics, and good food. Tuberculin may be used, beginning with a dose of 100,000 mg., and repeated once a fortnight in gradually increasing dosage; the temperature must be watched, any increase of symptoms, such as pain and fever after an inoculation, indicating that a negative phase has been produced by too big a dose.

Of local treatment, the most important part is to procure immobilization; also, the weight of the head and trunk must not be transmitted through the diseased *vertebræ*, and deformity

may be prevented or corrected to some extent by means of extension.

During the active stages of the disease the patient should be treated in the recumbent position. In cervical caries the spine is splinted by a jacket, preferably of plaster-of-paris, the head being immobilized by sand-bags or some form of box splint. When the disease has become quiescent the patient is allowed up in the plaster jacket with a bridle extension for the head; or a celluloid jacket, carried up to the occiput, mastoid processes, and jaw, may be used.

In the active stages of cervico-dorsal, dorsal, and lumbar caries the best apparatus is that perfected by Gauvain and named the "back-door splint"; for some cases in children a spinal board and jacket is preferable, and in cases without deformity a double Thomas hip-splint may be used. By these methods recumbency, immobility, and extension are ensured with apparatus which allows access to the diseased region. In the later stages the patient may get about in a jacket; plaster-of-paris reaching from skull to pelvis gives the best immobility, but necessitates expert technique for its application and renewal; failing this, a celluloid jacket is the most generally satisfactory form, being light, rigid, and durable, and causing but little disability to the patient.

In recent years, fixation of the spine by operative measures has given very satisfactory results, and it is likely to be used to an increasing extent. Albee's operation consists in splitting the spinous processes of the diseased *vertebræ* and of those above and below, and interposing between their lateral halves a bone-graft from the tibia; this forms a solid bridge which transmits the weight and gives absolute fixation. The operation shortens the recumbent period of treatment and hastens cure, but it necessitates special instruments and technique. A recent modification of this operation consists in the use of a boiled beef-bone graft, which is equally efficient; by this method interference with the patient's tibia is avoided and the operation is much simplified.

Direct operation to eradicate the disease is only possible in those rare cases where the transverse processes are alone attacked.

Abscesses should be dealt with, as a general rule, by aspiration, the trocar being introduced through the greatest possible thickness of healthy tissue at a point where the abscess is least likely to extend. This small operation

## SPINAL CARIES

has usually to be repeated more than once before a cure of the abscess results. It is safer not to inject drugs into the abscess cavity, as a rule, but if the pus is caseous 2-5 c.c. of a solution consisting of thymol 1 part, camphor 2 parts, sulphuric ether 3 parts may be introduced and left for a few days; it produces liquefaction of the pus and makes satisfactory aspiration possible.

Incision should be practised for retropharyngeal abscess in cervical caries, the pus being evacuated through an opening in the posterior triangle; such an abscess must on no account be opened in the pharynx. Open operation is also occasionally necessary for mediastinal abscess with increasing pressure symptoms. In rare instances paraplegia is due to the pressure of an intraspinal abscess, and in this class of cases alone laminectomy is indicated.

Sinuses are best dressed with gauze soaked in a 1-per-cent. emulsion of iodoform in liquid paraffin; the injection of a paste, consisting of bismuth subnitrate 1 part, sterile vaseline 2 parts, frequently leads to healing, which is aided by putting the patient in such a position that the opening the sinus is not at its lowest part. It may be necessary to counter-drain or open up sinuses, and to remove the sequestra which occasionally are present. Vaccines are of value for the secondary pyogenic infection.

Paraplegia is treated by absolute rest and the application of extension to the lower limbs by weight-and-pulley, counter-extension being obtained by fixing the patient's head or raising the foot of the bed. The cases which do not improve under this treatment are usually the result of transverse myelitis or fibrosis, and in such no lasting improvement will follow laminectomy.

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**SPINAL CORD, COMPRESSION OF**  
(see SPINAL CORD, TUMOURS OF; PARAPLEGIA, SPASTIC)

**SPINAL CORD, EMBOLISM AND THROMBOSIS OF** (see SPINAL CORD, LOCAL LESIONS OF; PARAPLEGIA, SPASTIC).

**SPINAL CORD, HÆMORRHAGE INTO**  
(see HÆMATOMYELIA; and SPINAL CORD, LOCAL LESIONS OF).

**SPINAL CORD, INFLAMMATION OF**  
(see MYELITIS).

**SPINAL CORD, INJURIES TO** (see SPINAL INJURIES).

## SPINAL CORD, LOCAL LESIONS OF

### SPINAL CORD, LOCAL LESIONS OF.

—The exact localization of spinal lesions has assumed a great importance in neurology, as not only do medical treatment and prognosis frequently depend on it, but the advance of surgical technique has made the successful removal of tumours, cysts, and other pathological conditions that involve the cord possible, provided that their position can be accurately diagnosed.

In localization we must determine both the region of the cord that is involved and the portions of its grey and white matter that are affected. As the symptoms on which reliance is placed in diagnosis are the evidences of functional disturbance that the patient presents, a knowledge of the functions of the different spinal tracts and columns of grey matter is essential.

**Diagnosis of extent of lesion.**—As it is from the cells of the *ventral horns* that the peripheral motor neurones take origin, lesions here produce palsies of the striated muscles as well as atrophy and changes in their electrical reactions. The reflex and automatic movements are paralysed, in addition to their voluntary contractions. In these respects the palsy is similar to that caused by injuries of the motor nerves.

As the *dorsal horns* receive the sensory root fibres, disease of these horns produces disturbances of sensation. Their form and distribution are variable and complicated. When the lesion extends to the tip of the horn and involves the entering fibres of one or more roots, we naturally find all forms of cutaneous sensation abolished or affected in the peripheral distributions of these roots; but since the various afferent impressions conveyed to the cord by the dorsal roots are rearranged in the grey matter of the dorsal horn, lesions here may produce partial or dissociated losses of sensation. A small focus near its base may, for instance, abolish pain and thermal sensibility alone on a restricted area of the surface of the body, or one of these forms of sensation only may be affected. Further, local lesions of the dorsal horns often cause sensory disturbances of a distribution quite unlike that met with in disease of the peripheral nerves, of the spinal roots, or of the white matter of the cord, since there is a regrouping of the afferent impressions in the grey matter according to their segmental origin in the limbs and trunk. Thus, a local lesion of one dorsal horn in the lower cervical segments may produce a

## SPINAL CORD, LOCAL LESIONS OF

dissociated anæsthesia on the hand alone, or on the hand and part of the forearm of the glove or gauntlet type, or, if it is situated in a higher segment, the shoulder and upper arm only may be anæsthetic.

The disturbances of function produced by *lesions of the white matter* are more simple. The most important of the motor bundles are the pyramidal tracts, which lie mainly in the lateral columns and terminate in the grey matter of the same side. Injury of one pyramidal tract affects those movements of the same side of the body that are innervated from the segments below the lesion; and as, in the adult, all voluntary impulses probably pass through the pyramidal fibres, a complete paralysis of voluntary movement results if the destruction is complete. There is apparently no segregation of fibres according to their function in the pyramidal bundles: in other words, those through which movements of the upper limbs can be excited are not anatomically separate in the cervical segments from those concerned in the movements of the legs, and partial lesions of the tracts do not consequently cause local palsies.

From the point of view of spinal localization, the most important of the ascending fibres are those that convey sensation. There are two main sensory paths in the cord. The one consists of fibres that spring from the cells of the dorsal horn and, after decussating in the dorsal commissure, run upwards in the opposite ventro-lateral column (Fig. 91). Afferent impressions that excite sensations of pain and temperature pass by this route, and local lesions of one dorsal horn, of the dorsal commissure, or of the ventro-lateral column may consequently produce loss of pain and thermal sensation only. Disease of a dorsal horn causes such analgesia and thermanæsthesia on a small area of the same side only; lesions in the commissure generally produce local bilateral disturbances, as the decussating fibres from both dorsal horns are likely to be involved; while injury of the ventro-lateral column affects sensation on the opposite side of the body only, and, since all the sensory fibres lie here in a compact bundle, there is generally analgesia and thermanæsthesia of the whole of the opposite side below a level that varies with the position of the lesion in the cord. As these crossed fibres decussate obliquely, the upper limit of the sensory loss is usually one or two dermatomes below that which corresponds to the injured seg-

ment. The sensory impressions conveyed by the sacral and lumbar roots do not, however, decussate below the first lumbar or twelfth dorsal segment, and lesions of the lumbo-sacral region therefore produce sensory disturbances on the same side only.

The second sensory path is formed by the root fibres that ascend through the dorsal column of the same side of the cord (Fig. 91). These fibres convey those impressions on which the sense of position and movement, the recognition of size, shape, weight, and vibration, and

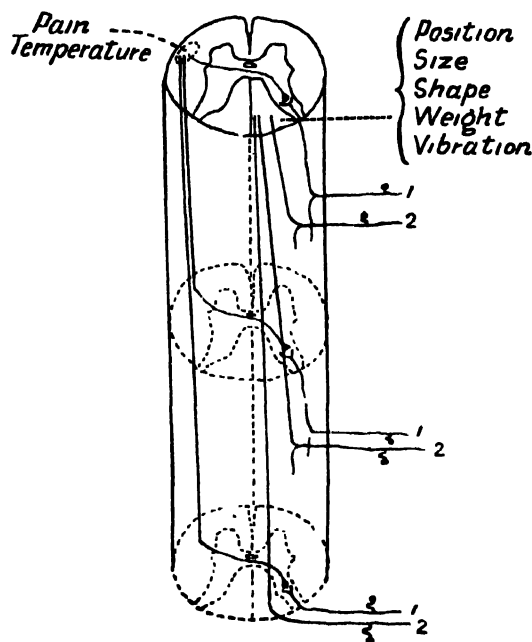


Fig. 91.- To show sensory conduction in spinal cord.

1. Root fibres which enter Lissauer's zone and terminate around cells in the dorsal horns; these send fibres into the opposite spino-thalamic tract.
2. Coarser root fibres which ascend in the dorsal column of the same side.

the discrimination of two or more simultaneous contacts depend. Tactile sensibility has a double path in the cord; it may ascend in either the dorsal column of the same side or the opposite ventro-lateral column.

A *unilateral spinal lesion* situated above the lumbo-sacral enlargement consequently produces characteristic sensory disturbances, pain and thermal sensibility being abolished on the opposite side of the body, while the sense of position and of movement of the limbs, and the recognition of size, shape, weight, and



## SPINAL CORD, LOCAL LESIONS OF

vibration, are lost on the same side; tactile sensibility, on the other hand, is little if at all affected. And on the side of the lesion there may be a narrow zone of anæsthesia owing to injury of the root fibres entering the dorsal horn; or there may be a zone of hyperæsthesia immediately above the level of the sensory loss. Further, as the pyramidal tract is generally interrupted too, there is usually a motor paresis of the spastic type on the same side and, if the ventral horn be damaged, an atrophic paralysis of the muscles innervated from the injured segments.

These symptoms produced by a unilateral spinal lesion are generally known as the *Brown-Séquard syndrome*, as they were first recognized by this distinguished neurologist. They are commonly met with in all types of spinal disease, but they are frequently less typical than as described here. A partial lesion situated in the ventral portion of the cord may cause only homolateral weakness with loss of pain and thermal sensations on the opposite side, while if it lies in its dorsal portion there may be only a disturbance of those forms of sensation that are conveyed by the dorsal column, and a spastic weakness of the same side.

### **Localization of the level of the lesion.**

—The exact level of the lesion in the cord can be determined by the extent of the motor, sensory, and reflex changes that it produces, and especially by identifying accurately the upper limits of these disturbances. It is usually on the sensory symptoms that localization is based, but the motor and reflex alterations frequently provide equally accurate information, and in employing them we have not to rely on the intelligent co-operation of the patient, which often fails us.

**Localization by sensory changes.**—This demands, in the first place, a careful determination of the upper level of the alterations in sensation, and, in the second, a knowledge of the distribution on the surface of the body of the dorsal spinal roots. (Plate 33.)

As tactile sensibility has a double path in the spinal cord, touch often escapes in incomplete lesions, or the alterations in it may be slight and easily overlooked. When a light stimulus is employed, as a wisp of cotton-wool or a camel's-hair brush, some loss or diminution at least can be usually detected, or the patient may recognize a difference between the sensations evoked on the affected and on the normal part; the wisp of wool

may tickle more, or appear heavier or more distinct, when in a series of contacts from below upwards the level that corresponds to the lesion is passed. Or there may be a zone of hypersensitiveness at this level. When the loss of tactile sensibility is complete the upper margin of the anæsthesia is generally lower than that of the analgesia.

Alterations in cutaneous pain sensibility are more easily elicited. A sharp steel pin should be employed, and the contacts should be only sufficiently heavy to produce pain or a stinging sensation on normal parts. Further, the examination should be always made from below upwards, and if tactile sensation is preserved the "covered point method" should be employed—that is, the point of the pin should rest against the pulp of the observer's finger so that both can be applied at the same time; otherwise the patient may recognize the pin by the smallness of its point, and respond though he may feel no pain. By this method it is easy to determine the exact level at which altered sensibility joins the normal. But it must be remembered that in unilateral spinal disease this is usually two or three segments lower than that which corresponds to the spinal lesion, owing to the oblique decussation in the cord of the pain-carrying fibres. Then a narrow band of hyperalgesia on the side of the lesion generally indicates the true level. At the upper limit of the analgesia there is frequently a zone of hyperalgesia, or increased sensitiveness to all stimuli; this valuable sign in localization is most easily demonstrated by drawing a pin-point lightly downwards till the patient winces or says that the stimulus gives him more pain.

The upper level of the thermanæsthesia, or alteration of thermal sensibility, generally corresponds with that of the analgesia. Its investigation demands considerable care, as errors are easy. Minimal stimuli only should be employed—that is, such as can be just distinctly recognized on normal parts—and changes should be recorded only when heat and cold cannot be distinguished, or when the sensations evoked by them become more definite above a certain level.

Those forms of sensation conducted by the dorsal columns are of less importance in localization. Occasionally loss of the sense of position or of movement at one or more joints in a limb indicates a level below which these forms of sensation are disturbed, but as the muscles and joints receive, as a rule, afferent

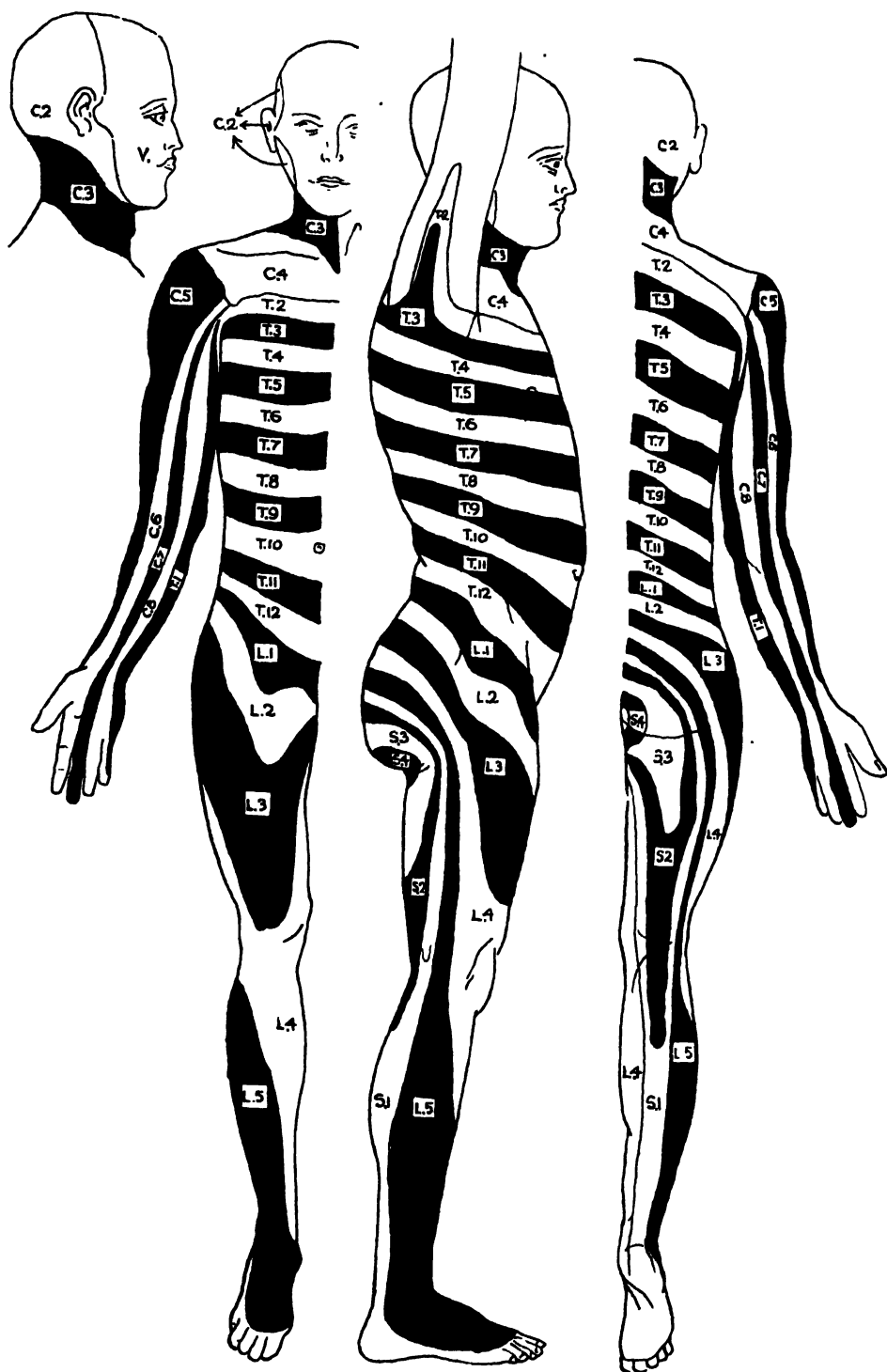


PLATE 33.—TO SHOW DISTRIBUTION OF THE DORSAL SPINAL ROOTS TO THE SKIN.



## SPINAL CORD, LOCAL LESIONS OF

fibres from more than one root, accurate localization is rarely possible by this means. In the appreciation of vibration, however, we have a valuable guide to the level of a lesion in the dorsal columns; the vibrating fork should be placed on a series of points from below upwards till the vibration is felt, or till it becomes more distinct; when the 9th dorsal segment, for instance, is injured, vibration is not felt, or not so plainly, below the level of the umbilicus.

We must also take into account various sensory symptoms of which the patient may tell us. As one or both dorsal roots that enter the affected segment may be irritated by the lesion, there may be pains referred to the sensory roots areas that correspond to them, or the patient may complain that at this level there are sensations of numbness, tingling, or constriction; a girdle sensation is often a valuable indication of the position of the lesion in the cord.

**Localization by motor disturbances** demands a knowledge of the radicular supply of the various muscles of the trunk and limbs. It is true that most muscles are polymeric—that is, receive motor fibres from two or more roots—but an accurate analysis of the paresis usually permits localization to within a segment or so. The paresis is often associated with wasting of those muscles supplied by the injured segment. A lesion of the 1st dorsal segment, for example, may produce wasting of certain of the small hand-muscles and spastic weakness of all the muscles of the trunk and lower limbs; if situated in the 7th cervical segment, it paralyzes the triceps but leaves the biceps unaffected; or if in the 1st sacral, the muscles of the calf and foot, while the anterior tibial group escapes.

Similarly a lesion of the 11th dorsal segment paralyzes the lower portions of the external obliques, so that on coughing the lateral portions of the abdominal walls in the region of the groins bulge, while the whole of the rectus abdominis contracts normally. And as the recti are innervated segmentally, lesions in the lower half of the dorsal cord produce paresis of certain segments of them only; one in the 9th dorsal segment paralyzes that portion of them below the umbilicus, and as the upper portion shortens while the paralysed part is passively stretched, when the patient tries to sit up the umbilicus is displaced upwards. The state of the intercostal muscles should be also observed, as it serves to indicate

the position of a lesion in any part of the dorsal cord. If the observer's fingers are placed in the intercostal spaces the muscles that form their floor can be felt to become tense and to rise on deep inspiration, but if they are paralysed no contraction can be felt and the floor of the space sinks in as the ribs separate. Since the intercostals are segmentally innervated, the highest level at which no contraction can be felt indicates directly the upper level of the spinal lesion.

**Localization by changes in the reflexes** is a valuable objective method, but as we cannot utilize reflexes that are subserved by every spinal segment it cannot always tell us the exact level of the lesion. A lesion in the 1st sacral segment abolishes the ankle-jerk; one in the 3rd or 4th lumbar abolishes the knee-jerk but exaggerates the ankle-jerk; and an injury of the 6th cervical leads to loss of the flexor jerk of the arm but the triceps or extensor jerk persists. The same rule holds for the cutaneous reflexes; the plantar cannot be elicited when the 1st sacral segment is damaged, or the cremasteric when the 1st lumbar is injured. In the abdominal reflexes we possess the most accurate guide of the position of a lesion in the lower half of the dorsal cord: if the skin of the abdomen is stroked from below upwards with the point of a pin, the level at which the reflex can be elicited can be determined; when a lesion involves the 10th dorsal segment no reflex can be obtained below the umbilicus; or if the 8th dorsal, contraction of the upper segment only of the abdominal wall, on each side of the xiphoid, can be evoked.

**Special symptoms.**—Certain symptoms dependent on injury of particular regions of the cord must be mentioned. Lesions of the 8th cervical and 1st dorsal segments paralyse the cervical sympathetic and produce ocular symptoms—enophthalmos, narrowing of the palpebral fissure, ptosis, and miosis—as well as vaso-motor and secretory disturbances on the same side of the head and neck: a flushing of the face, which is pronounced only at first, absence of sweat secretion, and diminution of tears, are the most prominent of these. As the vaso-motor fibres arise from the 2nd to the 4th dorsal segments, they alone may suffer, or there may be only ocular symptoms. Total transverse lesions of the lower part of the cervical enlargement lead to hypothermia with a body temperature of only 80° F. or so, bradycardia or a pulse-rate of 30–40 per minute, a great fall of blood-pressure, and

## SPINAL CORD, TUMOURS OF

diminished secretion of urine. This condition is invariably fatal.

One of the serious complications of injuries in the mid-dorsal region is paralytic distension of the intestines. It occurs chiefly with acute lesions of this region.

The nervous mechanism of the bladder and rectum are complicated. The bladder has two separate sources of innervation, from the upper lumbar segments through the hypogastric nerves, and by the sacral autonomic fibres that run in the pelvic nerves. Damage of either of these produces disturbances of micturition, but in man they do not differ essentially from those due to transverse lesions of any part of the cord. The general rule is that in an increasing transverse lesion of one of the higher segments the first symptom is difficulty and slowness in starting the flow of urine, which is followed, as the disease develops, by more and more complete retention, or overflow incontinence. Not infrequently, however, reflex incontinence supervenes; that is, the bladder is emptied periodically by a reflex contraction of its walls and relaxation of its sphincters, excited by the rising tension of the urine contained in it. Precipitant or urgent micturition is a common symptom of more chronic spinal paresis of the spastic type; in it the desire to micturate is followed quickly by an uncontrollable reflex evacuation.

Constipation, which becomes more and more obstinate as the disease advances, and faecal incontinence, especially if the stools are loose or if diarrhoea occurs, are the disturbances of defaecation produced by spinal lesions.

GORDON HOLMES.

**SPINAL CORD, TUMOURS OF.**-- New growths may interfere with the functions of the spinal cord either by compressing it or by invading it. Those which grow outside it and compress it produce the typical picture of a compression paraplegia, which is dealt with under PARAPLEGIA, SPASTIC, while the symptoms of those which originate within it, or spread to and infiltrate it, are distinguishable from those of other local lesions only by their course (see SPINAL CORD, LOCAL LESIONS OF).

Spinal tumours deserve, however, a separate description, as their diagnosis and localization often permit their surgical removal with brilliant results.

**Extramedullary tumours** may grow from the vertebral column, from the meninges, or

from the spinal roots. Sarcomata and carcinomata secondary to primary growths elsewhere, particularly in the breast, prostate, and rectum, are the most common vertebral tumours, but osteomata, fibromata, and tuberculomata also occur in this situation. Most of the intradural tumours are benign or relatively so; the most common are fibromata, lipomata, endotheliomata, psammomata, and sarcomata, but simple and hydatid cysts are also met with. Fibromata growing from the spinal roots as part of a general neuro-fibromatosis may also compress the cord. The sarcomata often spread over several segments and may encircle the cord, but the other forms of tumour are usually localized and easily removable.

The earliest symptom depends on the part that is first affected. This is often a spinal root, especially when the tumour grows from a vertebra, and then the first manifestation of the disease is radiating pain along the peripheral distribution of the root. A man, for instance, in whom an intradural tumour was recently removed from the lowest portion of the cervical enlargement, complained for months only of a shooting pain in the ulnar border of his left arm and little finger. In malignant growths of the vertebral column the roots of both sides may be involved, producing a girdle sensation or girdle pain. In these cases there is often, in addition, local pain and tenderness of the spine, which may direct attention to the disease, but it is rare unless the bones are actually invaded.

The next symptom is usually a feeling of stiffness and weakness of the legs and a tendency to tire easily in walking, which may increase slowly until gait or even standing is impossible. Rigidity generally develops *pari passu* with the weakness unless the latter increases rapidly, and we consequently have the clinical picture of a spastic paraplegia. Local muscle wastings, due to compression or invasion of the motor roots, may also develop, especially when the tumour lies over one of the enlargements; in the case just referred to, atrophy of the small muscles of the left hand was an early symptom.

As the spinal cord becomes more compressed the sphincters are affected, first only as a slowness in starting micturition, but later retention and probably overflow incontinence appear. Constipation is the rule, too, owing partly to the spinal lesion and partly to the inactivity that it induces; but as the control

## SPINAL CORD, TUMOURS OF

over the anal sphincter is deficient, incontinence is probable if diarrhoea occurs.

On examination we find the usual symptoms of a local spinal lesion. Sensation is generally affected later and less than motion, and, as one side of the cord may be more compressed than the other, a Brown-Séquard dissociation is not uncommon. When the tumour lies on the dorsal aspect of the cord the dorsal columns may be most damaged, producing loss of deep sensation and ataxia, while pain and thermal sensibility often suffer most when the tumour is ventral to the cord.

The knee-jerks and ankle-jerks are exaggerated if the lesion is above the lumbosacral enlargement, the plantar responses are of the extensor type, and the abdominal reflexes disappear if it is as high as the mid-dorsal region.

Neoplasms that involve the cauda equina or compress the lumbosacral enlargement are characterized by a special group of symptoms. As it is usually the dorsal roots that suffer first, severe radiating pains in the buttocks, perineum, and legs are the most prominent symptoms, and as the ventral roots are also involved the muscles waste rapidly, so that we have the condition of an atrophic palsy of the lower limbs with extensive disturbances of all forms of sensation and loss of all the reflexes. Sphincter paralysis with incontinence is an early feature of these cases.

Examination should, in the first place, be directed to the localization of the lesion that is, to the exact determination of its position in the spinal cord—and then to the diagnosis of its nature. Though we can easily localize the spinal lesion that the compression produces (see SPINAL CORD, LOCAL LESIONS OF), we can be less certain of its cause.

The chief distinguishing features of a compression paraplegia, in contrast to one due to an intraspinal lesion, are: (1) Its slowly progressive course. (2) The presence of referred pains due to involvement of one or more dorsal roots. (3) Symptoms indicating a diffuse transverse lesion of the cord rather than a localized injury of a limited portion of its cross-section. (4) A local deformity, rigidity, or tenderness of the spine to percussion is common in the case of extradural tumours that infiltrate the bone and are sometimes present with intradural tumours too. A radiographic examination should never be omitted. (5) Tumours, whether intra- or extradural, block the flow of cerebro-spinal fluid in the subarachnoid space, and, as a result of this, certain physical and

## SPINAL CURVATURE

chemical changes can generally be found in that obtained by lumbar puncture from below the level of the lesion. The fluid is generally yellow in colour (*xanthochromia*), contains a high percentage of albumin and may clot spontaneously, but has no excess of cells. This, the *loculation syndrome* of the fluid, is always suggestive of an extramedullary lesion.

But none of these signs is pathognomonic, and we are often in doubt whether the primary disease is intra- or extramedullary. Even in the latter case the symptoms are due to an affection of the cord. Further, we can rarely be certain of the nature of the lesion, whether it is a tumour, a cyst, or a thickening of the meninges. Consequently, in all cases in which there are reasonable grounds for suspecting a removable tumour, an exploratory laminectomy is indicated; the risk to life, or of further damage to the cord, is small in proper hands, and the possible benefit certainly outweighs any risk there may be.

But it is essential that accurate localization should precede surgical intervention.

**Intramedullary spinal tumours.**—The tumour that most commonly originates within the spinal cord is a glioma. It may be localized, or extensive and diffuse; in the latter case there is often a central softening of the tumour, producing a condition that resembles syringomyelia. Sarcomata, metastatic carcinomata, gummata, and tuberculous growths also occur.

The symptoms produced by these tumours are very similar to those due to other intramedullary lesions, but their onset is, as a rule, slower and more gradual. They can usually be distinguished from compressing tumours by the points emphasized above, but as this is not always possible it may be only on performing a laminectomy that a diagnosis can be made.

The treatment of intramedullary tumours is almost hopeless, except in the case of gummata, which often yield to energetic antisyphilitic measures. In two of my cases growths that could be shelled out were removed by operation, in one case with a favourable result; but this can rarely be expected. (GORDON HOLMES.)

**SPINAL CURVATURE.**—In this article Lateral Curvature, or Scoliosis, is considered, with Kyphosis and Lordosis; Angular Curvature is treated of under SPINAL CURVES.

**Etiology.** 1. Congenital scoliosis is a rare condition, and is usually due to one of the lumbar or cervical vertebrae being wedge-

## SPINAL CURVATURE

shaped; or it may be associated with congenital elevation of the scapula (Sprengel's shoulder).

2. Rickety curvature of the spine may arise before or after a child begins to walk; there is a diffuse curve of the whole spine with associated dorsal kyphosis.

3. Scoliosis may be secondary to many conditions which cause asymmetry: it results from torticollis, which may be due to several causes: it is also associated with any condition which produces unilateral limitation of respiration, such as phthisis or old empyema, or with loss of function of one arm; and it is very commonly secondary to shortening of one lower limb from any cause, such as dislocation of the hip, coxa vara, fracture, contracture of hip or knee, defective growth after infantile paralysis, and genu valgum.

4. Occasionally scoliosis results from injuries or diseases of the spine, especially fractures and tuberculosis, in association with angular or diffuse kyphosis.

5. Lateral curvature due to muscular causes may be (i) paralytic, resulting from anterior poliomyelitis, diphtheria, progressive muscular atrophy, Friedreich's ataxia; (ii) functional or spasmodic, from hysterical contraction; (iii) adolescent, from weakness of the muscles of the back in association with bad habits of sitting or standing.

*Adolescent scoliosis* is the most important variety, and is far commoner than any of the others; it usually begins between the ages of 10 and 20, and much more frequently affects girls than boys. The predisposing causes of its development are rapid growth and increase of the body-weight, and some focus of toxæmia, especially chronic infection of tonsils, pharynx, teeth, or lungs, and constipation. As a result of these factors the child becomes anæmic and debilitated, and there is general muscular atrophy and loss of tone. Either because of lassitude or on account of some trivial injury or inflammation of one of the lower limbs, the child develops a habit of standing with most of her weight on one foot. She may be accustomed to walk with her body crooked while carrying heavy weights. In other cases the deformity begins after some affection of lung, pleura, or chest-wall which causes asymmetrical breathing. Another common cause is a habit of sitting in a twisted attitude while writing at school; this is exaggerated by the use of a low desk and a seat without a back. Sometimes visual defects or unilateral deafness

may be the actual cause of the onset of the curvature.

**Pathology.**—In the slighter degrees there is no structural change, the deformity being purely postural; in more severe cases there is a single lateral curve of the dorsal and lumbar spine; in the most advanced cases there is a curve of the lumbar spine, usually convex to the left, and a curvature of the dorsal spine in the opposite direction, associated with rotation of the bodies of the vertebræ, so directed that the spinous processes are displaced towards the concavity of the lateral curves. The muscles and ligaments on the convex aspect of the curve are stretched and thinned, those on the concave aspect are shortened. The vertebræ and intervertebral discs become wedge-shaped, and such associated deformities as kyphosis, lordosis, genu valgum, and flat-foot are often present.

**Symptomatology.**—In the early stages there are no subjective symptoms, the deformity developing very insidiously. The child then begins to feel an aching pain in the back, in the hip, and along the course of the intercostal nerves. She is unduly fatigued after slight exercise, and also after sitting and standing without something to lean against; anæmia and other signs of toxæmia are present. Many cases, however, are brought for treatment because the right shoulder or the left hip is noticed to be "growing out."

On examination of a case of slight degree there is seen to be a single dorso-lumbar lateral curve, with cervico-dorsal kyphosis and sometimes slight lumbar lordosis; there is, however, no structural change, the deformity completely disappearing when the patient exerts herself to stand upright with the shoulders braced back.

In the next degree of severity there is a lateral curve, but early structural changes do not allow of its being completely corrected by voluntary effort.

In a more advanced type of case there is a lumbar curve, with rotation, but without any structural changes in the dorsal spine.

In the typical fully developed deformity there is a lumbar curve, having its convexity directed to the left in most cases, and a dorsal curve in the opposite direction, with rotation of the vertebræ in both regions; the lateral curvature can be demonstrated by palpating the line of the spinous processes, but its magnitude is masked by the fact that the rotation causes these processes to be displaced towards

## SPINAL CURVATURE

the concavity of the curves. The rotation is apparent to the surgeon on looking along the back from below, with the patient bending forwards; the thoracic wall on the side of the dorsal convexity is then seen to project more than the other side. In the regular type with left lumbar convexity the distance between the left arm and the trunk is increased owing to the presence of the "false waist," which corresponds with the dorsal concavity, and the left hip is prominent; the true waist, corresponding to the lumbar concavity, is deepened on the right side, and the lower margin of the thorax and the crest of the ilium are in close proximity. The left thorax is flattened behind and projects anteriorly, the right scapula is prominent and placed higher than the left one, the right shoulder is displaced backwards, and the right pectoral region is flattened. The back muscles in the concavity of the curves stand out prominently.

It is important in all cases to exclude shortening of one of the lower limbs: this may be done by measurement from the anterior superior spines to the internal malleoli, but it is more accurate to observe the relative position of the two iliac spines or of the highest points of the iliac crests, when the patient is standing upright. It is very common to find half an inch or less of shortening with no apparent cause. If the scoliosis is secondary to shortening, and non-structural, it disappears when the limbs are equalized by raising the foot on a sufficient number of thin books or other flat objects.

All causes of secondary scoliosis, such as asymmetrical vision, torticollis, and asymmetrical respiration, must be looked for, as well as sources of toxæmia, particularly in mouth and pharynx. The heart and lungs must carefully be examined, for active defects of these organs contraindicate treatment by energetic physical exercises.

**Diagnosis.**—In congenital scoliosis a wedge-shaped vertebra can only be diagnosed with certainty by means of X-rays; in congenital elevation of the scapula it will be observed that the affected bone is smaller than the one on the normal side, and it is situated nearer the middle line.

In rickety scoliosis other signs of the disease are present.

Early adolescent kypho-scoliosis is liable to be mistaken for *spinal caries*, and vice versa; the most important distinguishing signs of the latter disease are localized rigidity, and pain

on pressure on the lateral part of the affected vertebræ, and on jarring the spinal column; in all suspected cases a lateral X-ray photograph should be taken.

Curvature resulting from old *infantile paralysis* may be diagnosed by the history, and the effects of involvement of muscles in one or both of the lower limbs, in paralytic scoliosis there is a marked degree of deformity, as a rule.

**Prognosis.**—In adolescent curvature a cure is to be expected if there is no rotation; rotation deformity can never be completely rectified, but even in severe degrees the symptoms can usually be banished and the deformity can be diminished or masked by suitable exercises, manipulations, and jackets.

**Treatment.** Having excluded the presence of phthisis or other active disease which contraindicates energetic treatment, it is first essential to cure sources of toxæmia, such as septic teeth, tonsils, and adenoids, and constipation. The habits of standing, sitting, and walking must then be inquired into and corrected; a sufficiently high desk should be provided, and the girl should sit square in writing, on a seat which has a back support for the lumbar spine. A sufficiency of mental and physical rest must be arranged; it is, as a rule, better for the girl to remain at school, but she should lie supine on a flat surface for two hours after the midday meal every day; she should have plenty of fresh air, and a tonic containing iron is indicated.

In *adolescent scoliosis* the aim of curative treatment is so to develop the muscles that they correct the deformity and support the spine; this should be done by exercises rather than by instrumental supports. The degree of curvature and rotation must be noted at the commencement of treatment, and the case should be seen sufficiently often to check its progress. Spinal jackets should be reserved for those cases which deteriorate in spite of treatment by massage and exercises, and for severe degrees of deformity.

Physical exercises must be carried out under skilled supervision: symmetrical gymnastics are sufficient for early cases in which structural changes are absent.

For more advanced cases, special exercises have two main objects: (1) the encouragement of asymmetrical breathing, to develop the compressed side of the thorax and to diminish the expansion of the other side; (2) training the patient to put most of her weight on that



## SPINAL CURVATURE

lower limb which she has been in the habit of relieving of weight. Manipulations and massage must be carried out regularly, to increase the tone of the atrophied and stretched muscles, and gradually to correct the deformity of curvature and rotation.

In certain advanced cases, special forcible manipulations under anæsthesia, with the repeated application of plaster jackets, lead to considerable improvement, but for these expert knowledge is necessary.

The simplest apparatus aims at developing a habit of standing and walking which will gradually correct the deformity; for this purpose the general raising of one foot, raising one heel, or thickening one aspect of the boot sole is employed, so as to alter the line through which the weight is transmitted.

The main objects of jackets, which may be made of plaster-of-paris, celluloid, or poroplastic material with metal reinforcement, are to promote asymmetrical breathing by cutting large windows over the part of the thorax whose development is being encouraged, and to support the thorax in a vertical direction.

Even in severe cases, where correction of deformity is impossible and a supporting jacket is necessary, exercises should be persisted in, for they relieve the symptoms of pain and fatigue.

In *rickety scoliosis* the infant must be kept lying down, by means of a padded wooden board or tray of other material, the back being massaged and manipulated daily to correct the curvature gradually.

In the *active stage of infantile paralysis* the muscles must be treated by warmth, and during recovery massage and electrical treatment should be given by an expert. It is most important to prevent scoliosis by means of a jacket applied early and so designed as to relax the paralysed muscles and to keep the weight off the spine. In advanced cases of paralytic scoliosis a moulded jacket is applied in the position of greatest obtainable correction; by massage and exercises, unaffected muscles may be encouraged to replace the functions of paralysed ones, whereby pain will be relieved.

**KYPHOSIS.**—A deformity in which there is increased posterior convexity of the spinal column, in the form of an angular or a diffuse curve. *Rickety* kyphosis affects the dorso-lumbar region, and is treated on the same lines as rickety scoliosis. *Adolescent* kyphosis, "round shoulders," affects the cervico-dorsal

## SPINAL INJURIES

region, and is common in growing girls and young women; it is treated on the same lines as adolescent lateral curvature, by massage, gymnastics, and correction of faulty habits. *Paralytic* kyphosis, resulting usually from infantile paralysis, generally requires an instrumental support, but certain cases are suitable for bone-graft operations. It may be added that kyphosis is frequently seen as a result of disease of the vertebræ, especially tuberculosis, osteitis deformans, and osteo-arthritis. It also results from compression fractures.

**LORDOSIS.**—A condition in which the lumbar concavity of the spine is exaggerated. It may be associated with adolescent kyphosis; it also occurs in any circumstances in which the centre of gravity of the body is displaced forwards, such as pregnancy, the presence of fibroid tumours or excessive fat, etc., the shoulders being thrown back to preserve the balance of the body; or it may be compensatory to flexion of the hip, dislocation of the hip, etc. When pathological, it is corrected by curing the condition to which it is secondary.

(C. W. GORDON BRYAN.

**SPINAL INJURIES.**—Injuries of the spine naturally owe their importance more to the damage which may have been sustained by the cord and spinal nerves than to that of the bones and soft parts. Severe lesions of the spinal column, even involving fracture and displacement, are occasionally unaccompanied by gross injury to the cord, and, on the other hand, the cord often proves to be seriously damaged when signs of injury to the spinal column are inconspicuous. Apparently slight injuries, even when unaccompanied by cord symptoms, should not be too lightly regarded, as persistent and troublesome results may follow the laceration of muscles and ligaments, and neurasthenic symptoms not infrequently occur. Further, evidence of organic disease of the cord may develop later.

Spinal injuries are either simple or compound, most of those met with in civil life being of the former variety, while naturally all gunshot wounds are compound. The chief importance of this distinction, however, from the point of view of infection, does not lie so much in the integrity of the skin and muscles as in that of the dura mater. Usually, though not invariably, laceration of the spinal membranes is evidenced by the escape of cerebro-spinal fluid from the wound. This must always be regarded as a matter of grave prognostic

## SPINAL INJURIES

significance on account of the almost inevitable development of meningitis.

### INJURIES OF THE CORD

In a fracture-dislocation the cord may suffer only a slight contusion, or it may be sheared completely across. Between these extremes lie all grades of damage. In gunshot wounds direct injury by a bullet, shell fragment, or in-driven bone, may cause complete division, or, on the other hand, only a slight contusion may result from the impact of a spent missile upon the spinal column. Again, all grades of severity exist between these extremes. Further, in the case of gunshot wounds the cord may suffer "concussion" when a missile of high velocity impinges upon any part of a vertebra but does not directly damage the contents of the spinal canal. When injured in this manner the cord may suffer only slight "concussion" changes, or it may be completely disorganized at the level involved. These "concussion" changes, as described by Gordon Holmes, consist of (1) swelling, or complete disorganization of axis-cylinder processes and nerve-cells, (2) oedema, (3) multiple punctiform hemorrhages, and (4) cavity formation.

Wherever actual destruction of nerve elements has occurred, the changes are permanent and irrecoverable; in so far as the function of nerve elements is only in abeyance on account of oedema and other temporary disturbances, recovery may take place.

**Diagnosis.**—In the earliest stages it is impossible to tell, from clinical examination, whether the symptoms are due to destruction or to a temporary loss of function, so that in no given case can a reliable prognosis be made within the first few days, and usually not for a much longer period.

When the cord has suffered even a moderately severe damage the portion distal to the site of injury is deprived of its function for a longer or shorter period, the more caudally-placed segments suffering less than the more proximal. Thus, flaccid paralysis, with loss of reflexes, total abolition of sensation, and retention of urine occur, as a rule, even when the level of injury to the cord is situated high up in the thoracic or even in the cervical region. These symptoms indicate the stage of "spinal shock." If the level of cord injury is above the lumbar enlargement, the passing off of this stage is marked by return of tone in the flaccid muscles, by reappearance and exaggera-

tion of the deep reflexes, and by periodic reflex emptying of the bladder. This change in the neurological signs does not imply any recovery in the cord at the site of damage. On the contrary, other things being equal, the more severe the damage to the cord, and therefore the more complete the cutting off of inhibitory control coming from above, the more exaggerated will be these reflex phenomena in the cord below.

The persistence or reappearance of any of the forms of sensibility in the lower limbs provides definite evidence that the cord has not been completely divided. The occurrence of extensor as contrasted with flexor spasms in the lower limbs is also, according to Head and Riddoch, indicative of incomplete division of the cord.

It is to be remembered that hysterical paraplegia and other purely psychical symptoms may follow an injury to the spine. The chief points in the differential diagnosis are (1) that in hysterical paraplegia sphincter control is unaffected, while with severe organic lesions retention of urine is invariably present at first; (2) that whereas in organic cases the limbs are at first flaccid and the tendon reflexes abolished, in functional cases muscular tone is unchanged and the deep reflexes are exaggerated, (3) that in hysterical cases sensory disturbance may either be absent or, if present, take some characteristic hysterical form such as "stocking anaesthesia."

**Treatment.**—A patient suffering from a recent spinal injury must be gently handled, and moved only with the greatest care, lest further damage should be done. It is during the stage of spinal shock, too, that the tissues are most vulnerable and acute bedsores most likely to develop. Hence all possible precautions must be taken to avoid pressure, rubbing, and wetting of the skin. It is in this stage, too, that the bladder is most prone to become the seat of cystitis, so that the greatest care must be exercised in the use of the catheter. Not only must the instrument be sterile, but the penis must be cleansed with the most meticulous care. As soon as reflex emptying of the bladder can be established, the use of the catheter must be discontinued. The most potent factors in preventing the establishment of reflex evacuation are overdistension and sepsis. Any form of toxæmia, whether arising from the wound, from cystitis, from bedsores, or from broncho-pneumonia, exercises a depressing influence upon the reflex activity of

## SPINAL INJURIES

the spinal cord, and so delays or prevents the establishment of the automatic bladder.

Cystitis can only be effectually combated by the return of vesical function, whether voluntary or reflex. Mere drainage of the bladder does not, of itself, either prevent or cure cystitis. Nor can vesical lavage, or the administration of urinary antiseptics by the mouth, clear up cystitis in the absence of functional activity of the bladder, though these procedures are very useful in diminishing infection. Seeing that the vast majority of patients with spinal injuries succumb sooner or later to cystitis and pyelonephritis, the necessity for early and efficient treatment of the bladder cannot be overestimated.

**Bedsore.**—The prevention of this sequela of spinal-cord injuries is considered in the article on BEDSORES.

**The paralysed limbs.**—It must always be borne in mind that, during the time that the limbs are paralysed, deformities are liable to occur which may seriously interfere with their usefulness should recovery of power take place. Consequently, attention must be paid to the posture of the limbs, so as to prevent contractures and to keep the joints supple. Properly applied splinting is often valuable. The perfunctory use of electricity and massage is to be condemned. Spastic muscles do not require stimulation; on the contrary, everything possible should be done to avoid provoking muscular spasms. If massage is used as a means of improving the general nutrition of the paralysed limbs, it should be employed only with the greatest gentleness. When flaccid palsy exists, on the other hand, as in injuries to the cauda equina, massage and electrical treatment are necessary.

Spasticity provides one of the most difficult problems of treatment. Not only does it tend to obscure any voluntary power that may be present and lead to the development of deformities, but it often interferes with sleep and seriously undermines the patient's general health. By splinting, tenotomies, tendon transplantations, and sometimes by nerve section, a good deal may be done to diminish spasticity and so to relieve the patient of a continual source of distress.

As recovery gradually takes place in favourable cases, education of the previously paralysed limbs must be begun. Loss of function in the limbs is not only due to interference with motor paths, but may also depend largely upon loss of appreciation of the afferent im-

## SPINAL NERVES, LESIONS OF

pulses coming from the affected limbs. The patient has, during the time that his legs have been paralysed, forgotten how to walk, and must be taught. Thus, definitely directed educational exercises of the Frenkel type should be used, and the swimming-bath may be very advantageously employed.

It must also be remembered that psychopathic causes may underlie loss of function, and that, if present, they require to be dealt with on appropriate lines.

**Operative treatment.**—The question of laminectomy requires careful consideration. When the evidence of total destruction of the injured segments of the cord is sufficiently definite, the operation would clearly be useless. When, however, there is evidence, however slight, of conductivity through the injured part of the cord, an exploratory laminectomy is often advisable, since some remediable condition in the membranes or other structures surrounding the cord may exist which interferes with its recovery. Thus, operation may reveal a cord stretched over a bony prominence; compressed by a narrowing of the canal, or by a loculated collection of cerebro-spinal fluid; or constricted by scar tissue. In such circumstances, none of which can be foretold with any precision, operation is at least capable of putting the cord under more favourable conditions for such recovery as may be possible. These exploratory operations are only permissible in the absence of a septic wound.

PERCY SARGENT.

## SPINAL MENINGITIS (see PARAPLEGIA, SPASTIC).

### SPINAL NERVES, LESIONS OF. 1.

**Brachial plexus palsy.**—The nerve supply of the upper extremity may be damaged either by injury or by disease (i) of the primary nerves or ventral divisions of the spinal nerves between the intervertebral foramina and the plexus, (ii) of the plexus itself, (iii) of the nerve trunks below the plexus.

(i) **Lesions of the primary divisions above the plexus.**—The spinal nerves which take part in the formation of the brachial plexus consist mainly of the ventral branches of the 5th, 6th, 7th, and 8th cervical and 1st dorsal nerves.

The nerve supply to the upper extremity may vary either upwards or downwards in its origin from the spinal cord by as much as about half a root, according as to whether

## SPINAL NERVES, LESIONS OF

the plexus is of a prefixed or postfixed type. Thus, in a typical prefixed plexus there will be a large contribution from the 4th cervical nerve to join the 5th, innervating the deltoid and spinati muscles, and the 5th cervical nerve will be much larger than in the postfixed type. In addition, there will be a moderately large branch from the 7th cervical nerve to join the inner cord, forming the outer head of the ulnar nerve, and the 1st dorsal nerve will be smaller than in the postfixed type of plexus, and will receive no contribution from the 2nd dorsal.

The primary nerves may be damaged either (a) by a direct injury as by a stab or gunshot wound, neuritis from chill, pressure, involvement by growth, or osteo-arthritis of the vertebræ; or (b) by violent wrenching applied for forcible reduction of a dislocated shoulder under an anæsthetic, or traction on the upper extremity of infants during birth (one of the forms of obstetric paralysis), or wrenching of the limb by the clothing being caught in moving machinery. (c) Again, chronic pressure as in cervical ribs, or the pressure of an aneurysm, may damage the lower nerves leading to the plexus. In cases in which violence has been extreme the spinal nerves may be actually torn from the spinal cord. In such a case, when the 1st dorsal root has been damaged above the origin of the white ramus communicans to the cervical sympathetic, the pupil on that side becomes small, fails to dilate properly when shaded, and there is slight ptosis or drooping of the upper lid.

Injury involving the 5th and 6th cervical nerves, described by Erb in adults and by Duchenne in new-born infants, produces what is known as *Erb's palsy*, or *Erb-Duchenne paralysis*. In adults this type of paralysis occurs usually as the result of a fall from the height of a few feet upon the point of the shoulder, as in falling out of a cart. Such an accident depresses the shoulder and at the same time flexes the head towards the opposite side, thus forcibly stretching the uppermost and most oblique nerve to the plexus. Scarring may afterwards be found at the junction of the 5th and 6th nerves at the outer edge of the scalenus medius, though faradic tests always show the 5th cervical nerve to be the more seriously damaged of the two; indeed, this nerve may be ruptured within its sheath. Similar damage may occur as the result of gunshot or stab wounds. The paralysis involves mainly the deltoid, spinati, biceps, brachialis anticus, and supinator longus (brachio-radialis) muscles.

In a prefixed type of plexus there is also paralysis of the radial extensors of the wrist and the pronator radii teres; but in a post-fixed plexus these latter muscles escape, and there may still be some power of flexion by the biceps. No anæsthesia is detectable unless the 6th cervical nerve has been grossly damaged; this injury produces numbness and slight anæsthesia of the thumb and index finger.

The paralysed limb is adducted to the side by the unopposed pectoral, and rotated inwards by the subscapularis, while the forearm is extended owing to the paralysis of the flexors of the elbow. Gradually wasting of the deltoid, spinati, and biceps becomes apparent, the acromion process and head of the humerus become sharply prominent, while the humerus drops away from the glenoid cavity owing to the loss of tone in the paralysed muscles. This last phenomenon is, however, less obvious than in many cases of acute poliomyelitis.

The *prognosis* of these cases varies with the severity of the injury. The more severe cases never show any improvement, even with energetic massage and daily galvanism. If the electrical reactions a month after the injury suggest a complete nerve rupture—that is to say, if there is an entire loss of faradic irritability, with much-diminished and sluggish reaction to galvanism—the question of operation and an attempt at nerve suture may be considered. Several cases of obstetrical paralysis have been reported as much improved after this operation, but, on the whole, nerve-suture for injuries so near the spine is not successful.

Damage of the lower nerves which form the inner cord of the plexus may be produced by violence by which the arm is pulled downwards; paralysis of these nerves is sometimes known as *Klumpke's paralysis*, and the muscles involved are the intrinsic musculature of the hand and the flexors in the forearm, with the exception of the pronator radii teres and flexor carpi radialis, which receive their nerve supply from higher roots. Anæsthesia of the ulnar type, involving the inner border of the hand and the little finger, and of the 1st dorsal distribution along the inner border of the forearm, is usually pronounced; in post-fixed types the intercosto-humeral area in the upper arm may also suffer.

The congenital anomaly of an extra rib attached to the 7th cervical vertebra may be responsible in adolescence or early adult life

## SPINAL NERVES, LESIONS OF

for symptoms of pressure on the 1st dorsal nerve. (See CERVICAL RIB.)

(ii) **Injury to the plexus** itself may occur from direct wounding, by involvement in scar tissue, or from an acute neuritis. Generalized brachial neuritis occurs in adults as a result of a rheumatic infection. This may follow exposure to cold, or may be the sequel to a neurofibrositis affecting the shoulder-girdle muscles and fasciæ. Pyorrhœa, septic infections of various kinds, colitis, etc., are all possible agents of this form of neuritis. Direct extension of inflammation from a tuberculous pleurisy is a rare cause of brachial neuritis, but the most severe case I have ever seen was followed by very rapid pulmonary tuberculosis a few months later. The pain of brachial neuritis is intense and constant, and liable to exacerbations. It radiates from the shoulder to the fingers, which may become swollen and slightly stiff; and tender points may be noticeable, though they are more intermittent. The pain may be likened to that of the flesh being gnawed off the bone. Often worse at night, it may be controlled to a slight extent by antiphlogistic poultices or other forms of moist heat, and electrical heating pads. Methylsalicylate or menthol liniments may be employed, but massage must on no account be used. Fixation of the arm in a sling as a rule is not tolerated for long, and in a severe case daily administration of morphia or heroin may be necessary. The pain often lasts a month to six weeks. As in sciatica, the disease is a perineuritis only and the nerve-fibres are not damaged, no anæsthesia or wasting usually occurring, though adhesions in the shoulder-joint supervene in many cases, requiring subsequent breaking down by wrenching under an anæsthetic. It is only in exceptional cases that trophic lesions occur in the articulations of the fingers and wrist.

In civil practice direct injuries of the cords of the brachial plexus as they lie above the clavicle are rare, although during the War innumerable cases of gunshot wounds damaging one or more cords of the plexus in this region occurred. The commonest nerve trunk to be damaged is probably the posterior cord, causing paralysis of the deltoid, latissimus, triceps, and the remaining distribution of the musculo-spiral nerve. A portion of this trunk only may be damaged, the cord being made up of posterior branches from the primary nerves entering into the plexus.

Operations on the cords for gunshot injuries

in this region have not, as a rule, had very happy results, the scar tissue formed as the result of suppuration making accurate dissection almost impossible.

(iii) **Lesions of the nerve trunks** as they issue from the plexus are commonly produced as the result of pressure by the head of the humerus when the shoulder is dislocated. The head of the bone is usually dislocated downwards, and as a consequence the bone carries with it muscular attachments of the infra- and supra-spinatus muscles. In this way the suprascapular nerve supply to the infra-spinatus is forcibly stretched, causing paralysis of this muscle, while at the same time the upper motor branch of the circumflex nerve as it winds round the neck of the humerus is also violently stretched in a downward direction, causing paralysis of the deltoid muscle. Thus, *deltoid and infraspinatus paralysis* is the commonest form of paralysis following dislocated shoulder. Next most common, and sometimes additional to the above, is *musculo-spiral paralysis* from pressure by the head of the humerus upon this nerve in the axilla. Dropped wrist, paralysis of the triceps, supinator longus and brevis, with radial anæsthesia on the back of the thumb and outer part of the hand, give us the clue to this nerve lesion.

All the extensors of the wrist, thumb, and fingers will be paralysed in musculo-spiral palsy, and in addition slight weakness of flexion of the forearm may be noticeable, due not only to paralysis of the supinator longus, which is really a flexor, but to weakness of the brachialis anticus, which receives two nerve-branches from the musculo-spiral. There is radial anæsthesia for tactile, painful, and thermal stimuli, but not for pressure. The area involved is limited above by the wrist, and on the inner side by the cleft between the middle and third fingers, spreading downwards on the dorsum of the whole of the thumb and of the first phalanx of the index and middle fingers.

Other nerves which may be injured similarly by pressure of the head of the humerus after dislocation are the median, ulnar, and musculo-cutaneous. Any or all of these may be paralysed together.

*Musculo-cutaneous paralysis* is characterized by weakness and wasting of the biceps and brachialis anticus and, in a complete lesion of the nerve, by an area of anæsthesia along the front and outside of the forearm as far down as the wrist. The brachialis anticus is never

## SPINAL NERVES, LESIONS OF

completely paralysed, because of its nerve supply from the musculo-spiral. Flexion of the forearm also may be partly performed by the supinator longus.

*Paralysis of the median nerve* causes weakness of the flexors of the wrist, fingers, and thumb. The little finger, and to a less extent the third finger, can be flexed, though no flexion of the thumb and index is possible. Sensation is likely to be diminished on the outer side of the hand, thumb, and first two fingers. In cases of complete destruction there will be total anæsthesia to all forms, including pressure, on the palmar surface of the thumb and outer two fingers, and on the dorsum of the two peripheral phalanges.

*Palsy of the ulnar nerve* causes less obvious weakness of flexion of the fingers than do lesions of the median, flexion of all the fingers being possible, though that of the little finger is weak. There will, however, be flaccid paralysis of all the interossei and hypothenar muscles. In the forearm the flexor carpi ulnaris will be paralysed, so that in flexion of the wrist the hand deviates towards the radial side.

The electrical reactions in these cases of nerve injuries show marked changes, varying from slight diminution to faradism in the slighter cases, up to the complete reaction of degeneration. A frequent sequel of neuritis of the median and ulnar nerves is fixation of the interphalangeal joints by adhesions, unless great care is taken by daily passive movements to avoid their occurrence.

Palsy due to pressure on branches of these nerves by tight bandaging of the forearm for sprain or fracture may complicate the condition known as ischæmic myositis.

Paralysis of all three nerves, musculo-spiral, median, and ulnar, may be produced by pressure, that of the musculo-spiral being by far the commonest. Pressure of the head on the arm during sleep, or by the back of a wooden chair over which it is thrown, and especially the pressure of a crutch in the axilla, are fairly common causes of wrist-drop. Crutch palsy usually paralyses the triceps as well as the extensors of the wrist and fingers, owing to the compression of the nerve as it winds round the humerus being sufficiently high to involve the nerve supply to this muscle.

During anæsthesia, if the operating-table is not a narrow one, the musculo-spiral nerve may similarly be pressed upon. Severe musculo-spiral paralysis is commonly due to fracture of the humerus, the nerve being lacerated by

the sharp fragments of bone, or later it may be compressed by the formation of callus. In sleep palsy, or postanæsthetic paralysis, the triceps often escapes partially, as the pressure on the nerve is exerted rather lower down.

Pressure on the median nerve in the flexor muscles of the forearm is a somewhat rare occurrence, as is also pressure on the digital branches of the median in the palm of the hand by a round-handled umbrella or walking-stick. The ulnar nerve is rather more commonly affected than the median by pressure of a hard wooden desk upon the bent elbow, as when falling asleep at a writing-table. The ulnar nerve may also be injured at the elbow-joint by direct violence, as in fractures of the joint, and it is liable to slow chronic compression in its groove where it winds round the inner condyle by bony hypertrophy. This condition may necessitate lifting the nerve from its bed and bringing it in front of the inner condyle into the forearm.

*Birth palsy.*—Mention has already been made of a form of birth palsy described by Duchenne, in which pressure upon the 5th and 6th cervical nerves by a blunt hook or by the finger of the obstetrician in aiding delivery causes paralysis of the same group of muscles as described by Erb in adults, namely, the deltoid, biceps, brachialis anticus, supinators, etc. This paralysis is almost always unaccompanied by sensory changes, the damage really being done to the 5th cervical nerve rather than to the 6th. If the latter nerve is also seriously damaged there will be anæsthesia over the circumflex area and dorsum of thumb and index finger. Occasionally in birth palsy, as in adult cases, the lower cervical and 1st dorsal nerves may be damaged by stretching, in which case there is paralysis of the forearm and hand muscles proportional to the severity of the damage. As in Klumpke paralysis in the adult, due to stretching or rupture of the 1st dorsal and 8th cervical nerves, there may be sympathetic paralysis of the pupil if the damage is effected close to the spinal cord.

**2. Injury of the lumbo-sacral plexus** is rare, owing to its protected position, except as the result of gunshot injury. Pelvic tumours may compress portions of it, particularly the obturator nerve, producing paralysis of the adductors of the thigh, with anæsthesia on its inner and upper portion. Psoas abscess due to tuberculous caries of the lumbar vertebrae or of the ilium may set up sciatic neuritis and

## SPINAL NERVES, LESIONS OF

cause foot-drop, loss of the Achilles jerk, and anæsthesia of the foot; or the anterior crural nerve may be damaged, causing weakness of extension of the knee, loss of knee-jerk, and wasting of the front of the thigh.

Tuberculous disease of the sacrum may involve the sciatic nerve at its exit from the pelvis at the sciatic notch in the ilium. Wounds of the buttock by stab, and especially by gunshot injury, at any level from the buttock to the knee, are frequent causes of sciatic paralysis. The external and internal popliteal portions of the sciatic nerve, though usually bound up in one sheath to make the sciatic trunk, are really separate nerves, and may be described as such up to and above the sciatic notch. Indeed, sometimes the pyramidalis muscle passes between these two parts. Of the two the external popliteal nerve seems the more liable to injury.

Pressure is a rare cause of paralysis of the sciatic trunk, though it has been seen after a prolonged immobilization on a hard floor, as in coal-miners rendered unconscious after explosions of firedamp. Pressure on the external popliteal nerve where it winds round the neck of the fibula is a common accident, and is met with after anæsthesia, and in men whose work necessitates lying on a hard surface as in slating a roof.

**Treatment.**—The general lines of treatment of paralysis of the brachial plexus or its separate nerves are similar for all. If there is any indication of an active neuritis, as in brachial neuritis or sciatica, the limb must be rested and no massage given. When the paralysis is not due to an inflammatory lesion, but is the result of a pressure or wounding of a nerve, massage is usually advisable from the first, with daily electrical stimulation of the paralysed muscles by galvanism. A point of some importance is to prevent the paralysed muscles from being chronically overstretched. Wrist-drop due to musculo-spiral palsy may be obviated by the wearing of a light splint, so arranged as to keep the wrist and finger very slightly extended; it must, however, be taken off twice a day and the joints thoroughly moved, so as to avoid the risk of adhesions forming in the wrist and interphalangeal joints. This is a serious danger which may happen if forcible hyperextension is kept up continually for weeks, as was only too frequently done in army hospitals during the War. A better method than splinting

## SPLEEN, ENLARGEMENTS OF

the wrist is the use of an elastic glove extension such as that made for me by Mr. Henry Lewis, of 2 Westmoreland Street, W.1, which permits free use of the fingers and hand while maintaining passive extension of the wrist.

Similarly, foot-drop may be treated by an elastic extension, or spring boot to aid dorsiflexion. Paralysis of the deltoid and biceps, as in Erb's palsy, is more difficult to treat on these lines, but Fairbank's splint and others have been designed in order to keep the arm abducted at the shoulder and flexed at the elbow.

WILFRED HARRIS.

**"SPINE, RAILWAY"** (*see* NEUROSES, TRAUMATIC).

**SPINE, SPRAINS OF** (*see* SPRAINS).

**SPLANCHNOPTOSIS** (*see* VISCEROPTOSIS).

**SPLEEN, AMYLOID DISEASE OF** (*see* AMYLOID DISEASE).

**SPLEEN, ENLARGEMENTS OF.**—The spleen lies in the left hypochondrium, and in adults is not palpable below the margin of the ribs in normal conditions; in young children its lower margin may be felt during deep inspiration, or when the lower aperture of the thorax is widely opened, as in some cases of rickets, although there is no enlargement of the organ.

In clinical medicine it is rare for a splenic enlargement to be mistaken for some other tumour, unless the spleen is movable, or has been displaced to another part of the abdominal cavity. It is, on the other hand, extremely common for some other tumour to be diagnosed as an enlargement of the spleen. The most common error is to mistake a kidney tumour for an enlarged spleen, and in some instances the mistake is not only pardonable but, one may almost say, inevitable, the tumour being in the left hypochondrium, with a notched border toward the umbilicus. The points of distinction should be carefully remembered:—

1. A kidney tumour is apt to fill the loin, but a splenic tumour rarely does so.

2. A splenic tumour is apt to give an increased area of splenic dullness upwards, i.e. above the ninth rib in the posterior axillary line, and obliterate Traube's semilunar area of resonance; both of which are rare occurrences with kidney tumours.

3. The descending colon may be found lying in front of a kidney tumour, never in front of the spleen.

## SPLEEN, ENLARGEMENTS OF

4. The surface of a kidney tumour is commonly uneven; the surface of a large spleen commonly smooth.

Another disease in which splenic enlargement is often mistakenly diagnosed is tuberculous peritonitis, where the rolled-up omentum may form a mass which misleads the clinician.

The **causes** of enlargement of the spleen are so numerous that it is difficult to frame any classification which will be sufficiently comprehensive. The following includes all those commonly recognized :—

1. Enlargement due to **acute general infections**.

Typhoid fever.  
Infective endocarditis.  
Pyogenic septicæmia.  
Undulant fever.  
Occasionally other infective fevers.

2. Enlargement due to **chronic infective processes**.

Tuberculosis.  
Syphilis.  
Lymphadenoma.

3. Enlargement due to **protozoal infections**.

Malaria.  
Kala-azar.  
Possibly some others, e.g. Egyptian splenomegaly.

4. Enlargement due to **mechanical obstruction**.

Hepatic portal cirrhosis.  
Thrombosis of the splenic vein.  
Uncompensated cardiac disease.

5. Enlargement due to **new formations**.

Cysts, parasitic and non-parasitic.  
Malignant growths.  
Endothelioma (Gaucher's type of splenic anæmia).  
Amyloid degeneration.

6. Enlargement associated with **disorders of the blood**.

Idiopathic pernicious anæmia.  
Leukæmia.  
v. Jaksch's pseudo-leukæmic anæmia.  
Acholuric jaundice, congenital and acquired.

7. **Primary enlargement**.

Splenic anæmia.  
Splenic anæmia with hepatic cirrhosis (Banti's disease).  
Splenomegalic cirrhosis.

A few words in comment on this list will serve to point out the main difficulties of **diagnosis**. In the acute infective fevers and in malaria the diagnosis is usually easy; the

agglutination reactions, the results of blood-cultures, and the examination of films of the blood, give the requisite indications. Similarly leukæmia, v. Jaksch's anæmia, and pernicious anæmia can be diagnosed with certainty by blood-examination, and acholuric jaundice with splenomegaly has the characteristic fragility of the red blood-corpuscles to guide us.

In the majority of instances the diagnosis is difficult only in the more chronic enlargements. Of these, tuberculosis, syphilis, lymphadenoma, and splenic anæmia are the diseases in which distinction is most difficult. In the first three, the enlargement of the spleen is frequently associated with enlargement of the lymphatic glands, and a microscopic section of one of the glands will not seldom settle the point at once. The Wassermann reaction is sometimes also of service, but though it indicates the existence of a syphilitic infection, it does not necessarily follow that the splenic tumour is the result of that disease. The greatest difficulty, however, is that of splenic anæmia, firstly, because there is no general agreement as to what constitutes that disease, and hence there is a tendency to call by that name all splenic enlargements which are not obviously due to other causes; and secondly, because there is no certain method of diagnosing correctly between this disease and some other enlargements of the organ which probably belong to a different category; for example, it is difficult, and probably sometimes impossible, to say whether a splenic tumour with cirrhosis of the liver is an example of Banti's disease, or a mere secondary enlargement due to mechanical obstruction of the portal vein in the cirrhotic liver. The examination of the blood will give some aid on this point, for no case of splenic tumour in which leucopenia is definitely absent should be called splenic anæmia. It cannot be too strongly emphasized that the best means of avoiding error in the diagnosis of splenic tumours is an accurate and complete blood-examination.

Of the rarer forms of splenic tumour—cysts and new growths it is sufficient in this article to point out that they rarely simulate the other forms of splenic enlargement.

Two final observations must be made; one, that in one-third of all cases of pernicious anæmia the spleen is enlarged, occasionally to a very considerable degree; and the other, that when in a case of rickets the spleen is



## SPONDYLITIS DEFORMANS

palpable, it is often so by reason of its displacement rather than because it is enlarged, and that rickets, by itself, probably does not cause enlargement; in such patients some other explanation than splenomegaly should be sought.

HUGH THURSFIELD.

**SPLEEN, RUPTURE OF** (*see ABDOMINAL INJURIES*).

**SPLENIC ANÆMIA** (*see ANÆMIA*).

**SPLENIC ANÆMIA OF INFANCY (ANÆMIA PSEUDO-LEUKÆMICA INFANTUM)** (*see ANÆMIA*).

**SPLENOMEGALIC POLYCYTHÆMIA** (*see CYANOSIS*).

**SPLENOMEGALY** (*see SPLEEN, ENLARGEMENTS OF*).

**SPONDYLITIS DEFORMANS.**—A diffuse affection of the intervertebral joints, causing limitation of mobility and, sometimes, pronounced deformity of the spine.

**Etiology and pathology.**—Not uncommonly the spine is affected locally in rheumatoid arthritis and in osteo-arthritis. In rheumatoid arthritis the cervical spine is very often painful and stiff, but the condition usually remains confined to the cervical region, and there are slight, if any, X-ray changes. The lower thoracic and lumbar regions of the spine are prone to be affected by a localized osteo-arthritis, evidenced by lumbar pain and stiffness, and by lipping and osteophytes seen in radiograms. The term spondylitis deformans should be reserved for those cases in which, though other joints may be involved to a greater or less degree, the brunt of the disease falls on the spine, which is extensively, or perhaps universally, affected.

There are two important varieties of spondylitis deformans: (1) diffuse hypertrophic osteo-arthritis of the spine; (2) diffuse ankylosing arthropathy of the spine (*spondylitis chronica ankylopoietica*).

In the first variety the X-ray changes are obvious and symptoms comparatively slight. The disease begins in the intervertebral discs, which are absorbed and thinned, and lipping and exostoses are very evident; but the latter do not unite to link vertebra to vertebra. The smaller vertebral joints are similarly involved, but to a lesser degree, and, though mobility here also may be impaired by interlocking osteophytes, true ankylosis does not

occur. This type of the disease is found chiefly in advanced life.

The chronic ankylosing variety, which may attack younger subjects (ages 20–40), begins in the small intervertebral and costo-vertebral joints, and rapidly leads to fibrous, and eventually to bony, ankylosis. At the same time the spinal ligaments, especially the anterior, tend to become ossified, forming a continuous band of bone uniting two, three, or more vertebrae rigidly together. This ankylosing spondylitis has been further subdivided into two sub-classes—(a) the Marie-Strümpell class, in which the disease starts in the lumbar spine and spreads upwards, and the hips and shoulders are also involved; and (b) the Bechterew class, which begins in the cervical region and then spreads downwards. This classification is somewhat artificial, for there are many cases which are transitional between the two types. Von Bechterew believed the disease to be primarily a chronic meningitis, which, by compression of the nerve-roots, causes ascending degeneration in the spinal cord and also atrophy of the spinal muscles, with changes in the intervertebral joints secondary to the consequent muscular weakness. In these ankylosing cases the symptoms are severe and the X-ray changes slight. If the intervertebral discs are early involved, much bowing of the spine results, producing the picture familiar as the “aged rustic” of the stage. If ankylosis precedes disc-destruction there may be little or no deformity, but the spine becomes rigid from end to end (“poker-back”). In the ankylosing type the costo-vertebral joints are rigid and respiration becomes entirely diaphragmatic.

Sometimes an injury to the back seems to have initiated spondylitis deformans.

At least 80 per cent. of all the patients are males.

**Symptomatology and diagnosis.**—In most cases the diffuse pain in the back, with widespread rigidity and perhaps kyphosis, and the presence of root pains or paræsthesiæ, point to an obvious diagnosis, which may be confirmed by the X-rays examination (*see X-RAYS, DIAGNOSTIC USES OF*). Nerve-root pains are often severe and intractable, and may be prominent in the absence of narrowing of the intervertebral foramina; they may suggest an erroneous diagnosis, such as sciatica. The pains of spondylitis deformans often come on in crises and are particularly severe at night. Respiration may be extremely painful or, as already mentioned, may be purely diaphragmatic.

## SPRAINS

The **prognosis** is very unfavourable, and **treatment** is only symptomatic. We are powerless to arrest the course of the disease, which sooner or later leads to complete disablement. Any tendency to kyphosis must be combated at an early stage by a spinal poroplastic jacket, but efforts in this direction are not usually successful. The victims of the ankylosing type fall an easy prey to acute respiratory diseases.

MAURICE CASSIDY.

**SPOTTED FEVER** (see MENINGITIS).

**SPOTTED FEVER OF THE ROCKY MOUNTAINS** (see ROCKY MOUNTAINS FEVER).

**SPRAINS.**—When a joint is forced in any direction beyond its physiological range of movement and a rupture of one of its ligaments or of some fibres of a ligament occurs, the resulting injury is called a sprain. This is the true *joint-sprain*. Sometimes the ligament is torn from its attachment, taking with it a fragment of bone (*sprain-fracture*). Closely allied to it, and interfering similarly with the movement of a joint, is an injury to a muscle attachment in close vicinity to a joint. This is the so-called *muscle-sprain*.

**Pathology.**—When a ligament of a joint is damaged there is an immediate effusion of blood at the site of injury, rendered evident by a swelling at this point occurring very soon after the accident. As the bleeding ceases, an inflammatory reaction in the neighbourhood is set up, leading to much serous exudation; this increases the swelling and oedema already present. At the same time effusion, which may be hæmorrhagic, takes place into the joint. If the torn ends of the ligament are brought into apposition and kept there, healing soon takes place with recovery of normal joint-function. However, should a continuous distracting strain on the ligament be kept up by ill-advised use of the joint, healing will not ensue properly, and the injured region will remain a focus of a chronic inflammatory change. The localized swelling will remain, and there will be a chronic effusion into the joint with a corresponding disability.

In a muscle-sprain similar pathological phenomena occur: local swelling and bruising, with perhaps joint-effusion; and, in like manner, failure to keep the severed fibres in approximation will lead to persistent swelling and weakness in the affected muscle.

**Clinical features.**—True joint-sprains occur most frequently in the wrist, knee, or ankle. At the elbow, muscle-sprains are more often met with. There is a history of some wrench or sudden forcible movement of the joint which gives rise to severe pain, and may be associated with the sensation of something snapping or tearing. Immediately, the joint becomes useless. On examination, over the site of injury is seen a discoloured swelling, and all round, according to the time which has elapsed since the injury, there is oedema. The synovial cavity is more or less distended with fluid. It is easy to decide which ligament or muscle attachment is damaged, for pain is provoked by putting passively this ligament or muscle on the stretch. The site of the injury may be discovered also by the fact that it is the most tender point round the joint.

**Treatment.**—The principles are simple. The bleeding and effusion must be checked by applying pressure either by bandage or strapping. The injured ligament or muscle-insertion must be relaxed, and precautions taken to prevent the patient from inadvertently making a movement that would stretch the healing tear. Splints may be called for to effect this. In true sprains, active movements which do not put a strain upon the injured ligament should be instituted early.

When the condition has become chronic it is more difficult to treat. Here again steps must be taken to restrain such movements as continually subject the injured site to fresh trauma, and hence keep up the pathological process.

This limitation may be imposed by splints or properly applied strapping. It is in chronic cases that massage proves useful. Counter-irritation with Scott's ointment (ung. hydrargyri compositum) or ung. iodi may cause resolution of the nodule of inflamed fibrous tissue, and good results are sometimes obtained by ionization with chlorine or iodine ions, the cathode being applied to the site of disease and a maximum current of 2 ma. per square centimetre of active electrode being used. Ionization should last for twenty to thirty minutes daily.

### SPRAINS OF SPECIAL JOINTS

The **ankle-joint** is very commonly sprained. The injury is brought about by the foot being twisted so that the sole becomes inverted. The external lateral ligament is torn. A sprain-fracture may also occur. Swelling and

## SPRAINS

discoloration ensue around the external malleolus; soon oedema appears over a more extensive area. Maximum tenderness is elicited by pressure over the site of the injured ligament, and maximum pain by bending the foot inwards. There is fluid in the ankle-joint. Walking is exceedingly painful, if possible at all.

**Treatment.**—When seen early, elastic pressure should be applied by a firm figure-of-eight bandage over a layer of wool, which wool should appear as a fringe above and below the limits of the bandage. This last precaution is to provide against possible lymph obstruction caused by the hard margins of the bandage. The turns of the bandage should run from without inwards. A splint with footpiece is then applied. The bandage is taken off for half an hour twice daily and reapplied tightly. After about three days the patient may be allowed to walk in a boot to the outer border of the heel of which a wedge of leather has been attached; this keeps the foot a little everted and the injured ligament relaxed. Another help is to apply a strip of adhesive plaster about 2½ in. wide so as to limit inversion of the foot. The end of the strip is placed just below the internal malleolus with the foot slightly everted, and is then carried down under the sole and up the outer side of the leg for about two-thirds of the distance to the knee. It may be fixed by a piece of strapping round the leg just above the malleoli.

Another way to treat a sprained ankle is to apply strapping from the first. A strip is applied as described above, and then the ankle-joint is strapped in a figure-of-eight pattern in the ordinary manner. The strapping must be reapplied as soon as the oedema subsides.

A sprained ankle will soon get well if treated in this fashion, and massage will not be required. In chronic cases a little pad of strapping, made of half a dozen layers of plaster ¾ in. wide, sticky side outwards, should be applied to the site of the injury, as advised by Sir Robert Jones. Over this the ankle is strapped in figure-of-eight fashion. The wedge on the boot should not be omitted.

In the **knee-joint** a sprain is a far more serious accident than in the ankle. In the latter joint the strength of the articulation depends upon the shapes of the bones forming it. In the knee-joint strength resides solely in the various ligaments. If a ruptured liga-

ment does not heal satisfactorily and remains permanently lengthened, the stability of the joint is seriously impaired, and the condition is very difficult to remedy. Moreover, as in a simple sprain it is usually the internal lateral ligament which is injured, imperfect repair will predispose to dislocation of the internal semilunar cartilage to which it is attached. After a wrench the joint fills up with fluid, and great pain is complained of if an attempt to abduct the leg be made in the extended position. The bruising is over the ligament, and the tender spot is at some point in the course of the ligament or at its insertions. There is never locking of the joint in a simple sprain, as in dislocation of the internal semilunar cartilage, and in this latter condition the tender point is farther forwards, close to its anterior attachment.

**Treatment.**—Because imperfect repair is fraught with such dire possibilities to the joint, rest in bed with a back splint should be insisted upon for two weeks. At the same time the joint must be firmly bandaged over wool in the manner described for the corresponding injury to the ankle-joint.

Wasting of the quadriceps is likely to occur after this treatment unless guarded against. The patient must be instructed to make active contractions of his quadriceps muscle whenever, and as often as, he thinks of it. These contractions do not cause movement of the knee-joint, but they brace up the capsule, promote the absorption of joint-exudate, and diminish or prevent reflex wasting. At the end of two weeks the splint is removed. If the effusion into the joint has largely disappeared, the joint is strapped. The patient then sits on the side of the bed and practises movements at the knee-joint without putting any weight on the knee. In a few days he can walk on the joint. The strapping must be kept on for at least two weeks.

A very serious internal sprain of the knee-joint, due to severe trauma, is **rupture of one or both crucial ligaments**. The knee becomes exceedingly distended with exudate, and abnormal movements are possible. If the tibia can be moved forwards on the femur with the leg extended at the knee-joint, the anterior crucial ligament is ruptured; if, with the leg flexed at the knee-joint, the tibia can be moved backwards on the femur, the posterior crucial ligament is torn. Sometimes the spine of the tibia is detached with the anterior crucial ligament.

## SPRAINS

**Treatment.**—If detachment of the spine of the tibia causes an obstruction to complete extension, as it often does, the spine must be removed and the ligament sutured to the periosteum. This is the only indication for immediate operation in crucial-ligament injuries. In every case the limb should be immobilized in extension in a plaster case for six months; next, a back splint is worn during the day, but not at night, for three months. The patient can now be allowed to walk with the knee strapped. In old neglected cases the only hope of obtaining a stable joint is to make a new ligament from the fascia lata.

The **wrist-joint** is frequently sprained. There is sickening pain, and the joint becomes useless and swells. The maximum point of tenderness will be found over the affected ligament. It is very important not to mistake a Colles's fracture for a sprain. In a sprain there is an absence of the characteristic deformity and of the tender line at the site of the fracture. Elastic pressure by bandage and wool and a splint to keep the wrist dorsiflexed are the means to adopt in treatment.

At the **elbow-joint** muscle-sprains are much more common than true sprains. "Tennis elbow" and "golf elbow" are examples of them. The lesion may be close to the insertion of the triceps, brachialis anticus, or biceps, or to the origins of the common flexors or extensors from one or other condyle. They are easily diagnosed by the localized point of tenderness and the fact that active contraction of the injured muscle against resistance gives rise to pain, whereas other movements do not.

When the *triceps* tendon is injured there is tenderness with localized effusion just above its insertion into the olecranon process, and pain is felt here when the elbow is extended against resistance. In recent cases a sticking-plaster pad, made as described above, is fixed over the swelling with another piece of strapping, and an anterior splint is worn with the elbow nearly extended. It should be kept on for at least two weeks. When it is removed the condition should be treated as chronic cases are. A pad is placed over the site of the lesion as above, and a strip of 2½-in. strapping is wrapped round the upper arm a little above the elbow-joint. The hand is supported by a sling. The circular strip of strapping restrains movements of upper-arm muscles. Counter-irritation with Scott's dressing, iodine, or ionization may be required in this injury,

as it may be in the other muscle-sprains around the joints.

When the *biceps* insertion is sprained there is localized tenderness and pain on supination with the elbow flexed, as well as pain on flexion. Injury of the *brachialis anticus* tendon causes pain on active flexion of the joint only. Injuries of the *common flexor* or *extensor origins* are diagnosed by the same principles of examination. All these injuries are treated similarly to muscle-sprain of the triceps tendon.

Sprains of the **spine** are not uncommon injuries, and are not very difficult to diagnose. There may be rigidity, but it is only resistance to movements in the direction which puts the injured ligaments or muscle attachments on the stretch. Thus, in tearing of muscle attachments of the erector spinæ the spine will resist flexion, but if the patient is bent backwards passively over the surgeon's hand a considerable movement will be obtained. On the other hand, he cannot actively arch his back when lying down, because this will throw the injured muscle into contraction and give pain. In older or slighter cases the patient can bend forwards without pain, but the return to the erect posture is a painful movement. Sometimes lateral flexion to one side only is free. As elsewhere, there are tender points in spine sprains, and these lie to one side of the middle line.

The **treatment** of a sprain of the spine is relaxation of the sprained ligaments by putting the patient to bed with his back supported by a moderately soft pillow, and restriction of movements of the erector spinæ muscles by the application of overlapping pieces of strapping over a wide area of the back. Two weeks at least are necessary for bed treatment. In old cases adhesions may cause disability, and this may be removed by free and rather forcible movements under anæsthesia. The patient is first strongly curled up by extending the legs on the thighs, flexing the thighs on the abdomen, and pressing the head down on the chest. After this has been done he should be uncurled, and lateral movements of the spine similarly performed. Often he will wake up with his disability gone. In any case in which there is the slightest doubt of the diagnosis, such freeing of adhesions should never be done unless a skiagram has been taken previously; and, indeed, it is wise to make an X-ray examination before undertaking any forcible manipulation of the spine.

C. A. PANNETT.

## SPRENGEL'S DEFORMITY

**SPRENGEL'S DEFORMITY.**—Congenital elevation of the scapula. The scapula is raised, often small or altered in shape, and rotated so that its inferior angle approaches the spinal column. Its upper border forms a swelling in the supraclavicular fossa. Sometimes a bridge of bone or a rod of cartilage unites its vertebral border to the spine of the seventh cervical or first dorsal vertebra. The deformity may be bilateral, but is more often one-sided only and especially affects the right side. It is somewhat commoner in girls. Associated malformations are often found. Treatment, other than massage and exercises, is generally unnecessary, but surgical measures may assist movements by dividing shortened muscles and removing the hampering bridge of bone if it is present.

FREDERICK LANGMEAD.

**SPRUE** (*syn.* Psilosis).—A chronic disease occurring in tropical climates and characterized by a peculiar ulceration of the tongue and mouth and by changes in the gastro-intestinal tract, giving rise to chronic diarrhoea followed by anaemia and wasting.

**Etiology.**—Sprue mainly affects Europeans living in hot moist climates, especially on the sea coast, although cases are also met with in the uplands. Both sexes are attacked, but females more than males, and, as the disease usually only occurs after prolonged residence abroad, the incidence is most marked after the age of 35. Eurasians and natives of tropical climates are also affected, but not to the same extent as Europeans. Sprue is commonest in the Far East—China, Malaya, Japan, Sumatra and Java, but occurs also in Burmah, Ceylon, the Philippines, Australia, India, and the United States. Rare cases have been reported in Europe. Until recent years there was no general agreement as to the causation of sprue, and many theories to account for its origin were put forward from time to time. In 1901, however, Kohlberg of Java reported the isolation of *Monilia albicans* from the tissues of fatal cases, and since that time other observers—notably Castellani, Manson-Bahr, and Ashford of the United States—have published evidence in support of the view that sprue is due to an infection with one or more species of *Monilia*; this theory is the one most accepted at the present day.

**Pathology and morbid anatomy.**—The nature of the changes found in sprue show that it is an infection of a chronic type, the essential

## SPRUE

factors being a disturbed balance between the micro-organisms in the intestinal tract, with a preponderance of those of high carbohydrate-splitting power. The colon bacilli are said to be diminished in numbers; the stools are extremely acid and contain an undue amount of fat. *Monilia* may be obtained from scrapings of the tongue and from the stools.

The diminution in the size of the liver met with in the later stages is due to simple atrophy of the liver-cells, and is connected with the loss of the epithelial lining of the intestine and the resultant want of nutrition. The toxæmia which is also present is probably the result of an auto-intoxication following the altered conditions of the intestinal tract and the inability of the atrophied liver to deal with toxic substances elaborated in the intestines.

The morbid anatomical features met with in the alimentary tract are mainly of a chronic inflammatory nature, terminating in atrophic changes, and affecting, from above downwards, the mouth, pharynx, œsophagus, stomach, and intestines. In advanced cases there is much emaciation and muscular wasting, the skin is rough and dry, and eczema of the nates is not uncommon. Patchy pigmentation of the skin of forehead and cheeks also occurs.

**Blood changes.**—There is a definite anaemia in sprue, and the blood coagulation-time is increased. The red blood-corpuscles are decreased considerably in the advanced stages of the disease, while the hæmoglobin, although actually decreased, shows a relative increase. There is no actual but a relative leucocytosis, the proportion of white blood-corpuscles to the red being as 1 to 400; there is also a relative mononuclear increase and a diminution in the polymorphonuclear cells.

**Symptomatology.**—The incubation period is unknown; the onset of the disease being insidious and uncertain and the preliminary symptoms slight and evanescent. It is difficult for a patient to state definitely when his disease began. The first tangible evidence of the disease is shown in the mouth by the appearance of small vesicles on the tongue, which only cause a little temporary discomfort, soon disappear, and do not, as a rule, attract much attention.

After a short time—perhaps ten days or a fortnight—the vesicles reappear and disappear again, but last longer at their second appearance. Either then, but more frequently on the next occasion, they last much longer and

## SPRUE

break down into small aphthous ulcers, the irritation and discomfort of the vesicles being then replaced by an acute burning and stinging pain. At the same time the whole tongue is reddened and looks smaller than normal; its usual roughness is lost, and it appears smooth, dry, and glistening. Shallow ulcers with rugged edges may be seen on the edges and tip, and the superficial epithelium of the tongue and mouth peels off in flakes. There is accumulation of a glairy mucus in the fauces. Owing to the state of the mouth and tongue the patient finds that he cannot take food, except of the blandest character; such articles as pepper, vinegar, pickles, sauces, curries, etc., cause the most acute pain. For the same reason he cannot smoke.

This condition of the mouth may improve for a time and then return after a short interval with greater intensity; on the other hand, there may be a comparatively long interval with an absence of symptoms. These signs and symptoms may last for only two or three weeks, or may be prolonged with remission for several months or even years; during this time, except for the condition of the mouth and a feeling of lassitude, there may be nothing in the patient's condition to attract attention, except possibly some tenderness and enlargement of the liver. This is the *primary stage* of sprue.

Following the mouth changes, the œsophagus is affected, so that burning and choking sensations are experienced when food is swallowed; at the same time or very shortly afterwards the patient suffers from fermentative dyspepsia and some irregularity of the bowels. Again after an interval, which may be short or long, there is the onset of diarrhoea, the so-called *preliminary diarrhoea* of sprue. In this the bowels practically always move in the early morning, generally between 4 and 6 A.M., when two or three loose, copious, dark motions are passed, after which the patient feels much relieved, generally falls asleep again, and gets up in the morning feeling better, with a good appetite for breakfast. The stage of preliminary diarrhoea lasts ten days or a fortnight and then subsides. Afterwards the patient's health, except for the mouth symptoms, may remain apparently normal for weeks or months; or after a very short interval, the most characteristic feature of the disease, the *secondary diarrhoea*, sets in. Six or eight motions are then passed in the twenty-four hours, with greater frequency

in the early morning but not confined to that time as in the preliminary diarrhoea. The stools are very copious, loose, pultaceous, frothy, of a dirty-white colour, very acid, and have a sour, offensive smell, particularly if the patient has been taking a meat diet.

Accompanying the diarrhoea there are abdominal discomfort, tenderness on palpation of the abdomen, and flatulent dyspepsia: the abdomen is distended, and the appetite is capricious or lost. Like the earlier symptoms, the diarrhoea may improve for a time, the stools being reduced to one or two of an improved character in the twenty-four hours, and all the other symptoms ameliorated, or the diarrhoea may continue for many months. Failure of nutrition first becomes noticeable at this stage: the patient loses weight and energy, and the muscles waste, especially those of the arms and legs. At this time, too, the decrease in the size of the liver may first be observed and the patient shows signs of toxæmia. He is weak and unable to undertake any mental or physical exertion and his pulse becomes rapid, sometimes intermittent, and always of low tension. As the disease advances, all the symptoms increase in severity; there is much emaciation, the lips are cracked, dry eczema is present on the nates, and bedsores may form. In cases ending fatally there are increasing diarrhoea, prostration, and finally extreme exhaustion, consciousness usually being maintained until the last. As a rule, fever is absent throughout the course of sprue, although slight elevations of temperature may occur at irregular intervals and last for a day or two. The disease does not, however, always follow the course just described, as even in untreated cases recovery may take place after some years, the symptoms subsiding and the disease appearing to become latent; although the patient always remains liable to gastrointestinal disturbance brought on by very slight causes, there may never be any return of the characteristic symptoms. Further, as Carnegie Brown points out, in any one of the stages of the disease some of the symptoms may be suppressed, giving greater prominence to others, the mouth symptom being most prominent in one case and the symptoms of diarrhoea in another, so that some writers have described mouth sprue and intestinal sprue, although these are in fact only phases of one disease.

**Diagnosis.**—A well-marked case of sprue showing the symptoms and signs which have

## SPRUE

been described should present no difficulty in diagnosis; but in the earlier or later recurrent stages the diagnosis may be in doubt. In the earlier stages irregular diarrhoea in the early morning with coincident dyspepsia and mouth symptoms, and in the later the peculiar chronic diarrhoea and wasting, in a patient who lives or has lived in the tropics, should make the diagnosis clear.

**Differential diagnosis.** Sprue may have to be distinguished from the following conditions:-

1. *Stomatitis*.—The peculiar character of the mouth symptoms and stools in sprue, and the fact that in this disease the morbid conditions are not relieved by the removal of irritating factors such as excessive smoking, should enable the diagnosis to be made.

2. *Hill diarrhoea*.—In this affection the mouth symptoms of sprue are absent, and the disease usually occurs in high altitudes and is relieved by a return to the plains.

3. *Chronic dysentery*.—There are no characteristic mouth symptoms, the stools often contain blood and mucus, and they are not white in colour.

4. *Pellagra*.—The later stages of this disease might possibly be mistaken for sprue, but the history and earlier symptoms of the two diseases are entirely different.

5. *Pernicious anæmia*.—Although this disease might be mistaken for sprue owing to the similarity of certain of its symptoms, there is never in pernicious anæmia the wasting met with in this condition, and the blood-picture of the two diseases is different.

6. *Chronic pancreatic disease*.—The diagnosis in this case is difficult, as chronic disease of the pancreas may be prolonged and subject to remissions as in sprue. There are, however, usually no mouth symptoms, the liver is not decreased in size, and a tumour may possibly be detected in the region of the pancreas.

**Treatment.**—Treatment at present is mainly dietetic, and drugs hold but a secondary place. Mention must be made, however, of the observations of Ashford on the use of *Monilia vaccines*. Carl Michel, for Ashford, has reported 62 patients treated by autogenous *Monilia vaccines*, of whom 49 were cured, 12 improved, and only 1 died. The vaccine treatment requires the test of further experience before it can finally be accepted as a routine method, but it is well worthy of further trial. There is no specific drug treatment. Begg of Hankow introduced the administra-

tion of yellow santonin, for which he claimed considerable success; it is of no use, however, in the later stages of the disease.

The various symptoms or complications which may occur during the course of the disease must be treated on general principles. Small doses of opium may be used if the diarrhoea becomes acute, and act well. The mouth condition needs mouth-washes or paints, such as boric acid and glycerin, peroxide of hydrogen, etc.; cocaine may be necessary before the mouth is treated. Daily hot pads to the abdomen are recommended by Cantlie. A patient who is fit to travel and for whom a suitable dietary on the voyage can be arranged should, as a rule, be sent to Europe, except in the colder months of the year.

**Dietetic treatment.**—Various diets are employed in sprue, and of these the most useful are (1) the milk, (2) the meat, (3) the fruit, and (4) the mixed milk and fruit diets. It should be premised that with any diet the treatment should be preceded by a preliminary dose of castor oil, and the patient confined to bed for the first fortnight, even although he has been up and about before treatment is begun. He should also be weighed, and thereafter this should be done once weekly. Alcohol should be prohibited throughout the treatment, and smoking only permitted in great moderation. Although sprue is by no means a hopeless or intractable disease if suitably treated, successful treatment requires tact, discretion, and interest on the part of the medical man, and much patience and a ready acquiescence on the part of the patient.

**Milk diet.**—This diet is alike the simplest and most satisfactory, and has given excellent results, but it necessitates a sufficient supply of fresh cow's milk of good quality—a condition which cannot always be fulfilled in the tropics. All food except milk must be prohibited, and the amount of milk and the times of administration must be written out definitely and the instructions strictly adhered to, if success is to be attained. The method advocated by Carnegie Brown is the best, and has been used by the writer with success. The first stage of treatment (twenty-four days) is divided into six periods of four days each, the amount of milk in each period being progressively increased. During the first four days 60 oz. of milk are taken, divided into eight feeds, the amount being increased to 70, 80, 90, 100, and 110 oz. in each successive four-day period. The milk must not be

## SPRUE

sterilized or boiled, but in cold weather may be warmed; it should be taken slowly, either sipped, taken in a teaspoon, or through a glass tube. If the patient cannot tolerate milk in its ordinary form, it may be heated or diluted with an alkaline mineral water or barley-water. Citrate of soda, 1–2 gr. to the ounce, may be added with advantage.

The patient loses weight for the first week on this diet, but begins to gain after the tenth day and, if the treatment is successful, regularly gains in weight each week; at the same time all the symptoms ameliorate, the mouth loses its soreness, the flatulent dyspepsia improves, and the stools become less frequent and more normal in character. It is well to continue the plain milk diet for a month or six weeks, and, if the patient can take it, the amount of milk can be progressively increased to as much as 140 oz. per diem after the first twenty-four days. When this is done the number of meals should be increased rather than the amount taken at one time. At the end of a month or six weeks one egg beaten up in milk may be given, and the number increased until four are taken in the twenty-four hours, and, at the same time, a cup of weak China tea with milk and sugar, if liked, may be allowed twice daily.

Chicken broth, Benger's food, lightly boiled or poached egg, toast, pounded fish, and chicken can successively be added to the diet until the patient is taking fish, eggs, chicken, milk, custards, potatoes, and farinaceous food in small quantities; but the darker meats, most vegetables, highly spiced foods, curries, iced drinks, and alcohol must be avoided.

*Meat diet* can be resorted to when a patient cannot take milk or it cannot be procured. Fresh beef or mutton should be selected, and the diet begun with 2 lb. of beef, which should be minced finely with 2 oz. of suet, divided into six equal portions, and cooked in a buttered saucepan over a bright fire until the red tinge of the meat just disappears. A portion should be taken at 7 A.M., 10 A.M., 1 P.M., 4 P.M., 7 P.M., and 10 P.M.; half a pint of plain hot water, toast-water, or rice-water may be sipped before each meal. China tea, as in the milk diet, or with lemon, may be taken twice daily. The quantity of meat may have to be adjusted to suit the individual capacity of patients, but 2 lb. in the twenty-four hours may be regarded as the standard. After eight to ten days of the plain meat, the diet can be modified by replacing one of

## SPUTUM, VARIETIES OF

the morning feeds by a lightly boiled egg, and one of the evening portions by some light white fish; chicken may be subsequently substituted for a midday portion, and a banana or some grapes added.

No carbohydrates should be permitted, but after another week rusks or biscuits may be given, and the diet gradually brought to conformity with an ordinary invalid diet. In very advanced cases the treatment by a meat diet may have to be commenced with raw-meat juice.

*Fruit diet.* A diet of fresh fruit as a treatment for sprue was apparently first used, in Java, by van der Burg, who recorded great success with it. The fruits which are most useful are bananas, strawberries, and grapes; other fruits have also been employed. The general details of treatment should be carried out as in a milk diet. Plain water or toast-water is necessary as a drink if fruit is used to the exclusion of other food.

One medium-sized banana may be given every two hours and the number gradually increased until it reaches fifteen to eighteen per diem; afterwards twenty-four or more may be given. This diet can be continued for a month or six weeks, and is then gradually modified by the addition of other foods as in the case of the milk diet. Strawberries may constitute the sole diet, or may be combined with milk after the first eight days, should the patient's progress and condition allow it. This combination is perhaps more successful in treatment than when the diet consists of strawberries alone.

OLIVER ROBINSON.

## SPUTUM, EXAMINATION OF (see BACTERIOLOGY AND PATHOLOGY, CLINICAL).

**SPUTUM, VARIETIES OF** (see PLATE 3, Vol. I, facing p. 145).—The diagnostic value of a clinical and microscopic examination of the sputum need not be dilated upon. In this article the chief characteristics of the sputa in various respiratory diseases are summarized. For details of pathological methods of examination the reader is referred to BACTERIOLOGY AND PATHOLOGY, CLINICAL.

1. **Bronchitis.**—The sputum in the early stages is scanty, clear, and viscid, and consists chiefly of mucus. Later it becomes yellow or yellowish-green in colour from the presence of muco-pus, loses its tenacity, and is brought up in greater amount and with greater ease.



## SPUTUM, VARIETIES OF

Towards the end of an attack, when it again diminishes in amount, though still mucopurulent it is less fluid and more difficult to expel. In town-dwellers carbon particles may render it blackish, especially in the early and late stages, when its amount is not great.

Occasionally in bronchitis there may be a persistent *bronchorrhœa* of thin serous fluid. Another variety is thin, greyish, and foetid, and separates into layers like those of the sputum of bronchiectasis (*see below*). In *purulent bronchitis* the sputum may consist almost wholly of pus. This form was frequently met with in the influenza epidemic of 1918-19. In *fibrinous bronchitis* seaweed-like casts of tubes may be expelled; they are usually accompanied by some blood and mucus, and when unrolled the main stem may be found to be as large as the little finger. These casts consist mainly of mucin. Fibrinous bronchitis is not the only condition in which bronchial casts are expectorated. Membranous casts may be coughed up in diphtheria, and fibrinous casts in chronic heart disease, in pulmonary tuberculosis, and after aspiration of pleural fluid. In pneumonia and in pneumococcal bronchitis casts of the smaller bronchi are not uncommon, and large casts are occasional. A rare membranous cast is that of aspergillosis.

2. **Phthisis.**—Considerable involvement of the lung may be accompanied by very little or no expectoration. Usually the early sputum is scanty and mucoid, and has been likened to sago, this appearance being due to degenerated alveolar cells. Small grey or greenish-grey foci are often seen quite early. Later, the sputum is more abundant and purulent. With excavation the characteristic "nummular" form makes its appearance, each portion settling at the bottom of the spittoon in a coin-shaped mass, greyish-green in colour. It sinks if coughed into water or antiseptic solution, and in it may be detected foci of caseous material. The sputum of phthisis is often blood-tinged, apart from more considerable hæmoptysis.

Microscopic examination reveals elastic tissue, blood- and pus-cells, degenerated epithelial cells, hæmatoidin and cholesterol crystals, tubercle bacilli, the micro-organisms of secondary infection, and sometimes fungi. The elastic tissue may be derived from bronchi, blood-vessels, or alveoli. Most characteristic is the elastic tissue of the alveoli, which is branched and may outline the arrangement of the alveolar cells.

Occasionally calcareous masses are expectorated.

3. **Bronchiectasis.**—The sputum in bronchiectasis is greyish or brownish in colour, fluid, offensive in odour and often foetid. When poured into a conical glass it separates into three layers, a supernatant clear mucoid layer capped by a brownish froth, an intermediate deeper layer of muco-pus, and a granular sediment of pus and debris. It is occasionally blood-stained. In the sediment there are little pellets known as Traube's plugs, consisting of pus and epithelial cells and fatty-acid crystals. Microscopically, pus-cells, epithelial cells, and sometimes elastic fibres and blood-cells are seen. Fatty-acid crystals may abound, and cholesterol and hæmatoidin crystals are very frequent. The foetid odour is in great part due to butyric and valeric acids.

4. The sputum of **asthma** is distinctive. Early in an attack it is very scanty and brought up with great difficulty, and is then in the form of translucent, gelatinous pellets—the "perles" of Laennec. When these are unravelled they are found to consist of mucinous casts of small bronchi, twisted in a spiral fashion (Curschmann's spirals). Entangled in the mucin are leucocytes, chiefly eosinophils. Some of the spirals are merely twisted columns of mucin, others have a central clear core about which is coiled a skein of mucin fibrils. In many cases pointed diamond-shaped crystals (Charcot-Leyden crystals) are seen. After a few days the sputum loses its special features and resembles that of bronchitis.

5. In **pneumonia** the sputum is at first scanty and very tenacious, so that it adheres to the bottom of the spittoon. It is mixed with blood, which is red at first, but gradually darkens to a characteristic "rusty" colour. Sometimes unaltered blood is present in sufficient amount to give the sputum a reddish tinge throughout the disease, and blood alone may be coughed up at the onset. Occasionally the sputum resembles prune juice. Microscopically, epithelial cells, leucocytes, and red blood-cells are seen in various stages of degeneration. Other constituents are fibrinous casts of the bronchi, cellular moulds of the alveoli, and hæmatoidin crystals. The micro-organisms vary with the nature of the pneumonia. In *influenzal pneumonia* the sputum may be purulent and contain a considerable amount of blood. In *broncho-pneumonia* the sputum varies from muco-pus to almost pure pus, according to the nature of the infection.

## SPUTUM, VARIETIES OF

6. In **pulmonary œdema** a thin, frothy fluid, like soapy water, may be expectorated. It is often blood-tinged.

7. The sputum of **pneumonoconiosis** depends upon the variety of dust which has produced the disease. It is usually muco-purulent and abundant. In *anthracosis* it is very dark in colour from the inclusion of carbon particles. Many of these are seen to be within alveolar epithelial cells. In *siderosis* it is stained reddish from ferric oxide. In *silicosis* it may contain angular particles of silica. Workers in *aniline dyes* may expectorate pigmented sputum; for instance, bright blue in the case of those who work with methylene-blue. The sputum of millers and bakers may include doughy masses, and that of cotton-mill operatives, fibres.

8. Almost pure pus may be coughed up in cases of **pulmonary abscess**, or when extrapulmonary collections of pus, such as empyema, communicate with the air-passages. The odour is often fœtid, but seldom so offensive as that of the sputum of pulmonary gangrene or bronchiectasis.

9. The sputum of **pulmonary gangrene** is very characteristic. It is extremely fœtid and is usually abundant. It separates into three layers. Below is a sediment, greenish-brown in colour, and sometimes containing large fragments of gangrenous lung tissue. The intermediate layer is turbid but fluid, and greenish or brownish in colour, and above it is a thick cap of froth. Microscopically, abundant evidence of destruction of the lung is seen. There are numerous elastic fibres, granular debris, pigment, fatty-acid crystals, and micro-organisms, including leptothrix. Blood is often present, but is usually greatly altered.

10. In **new growth of the lung** the sputum may resemble prune juice from the presence of altered blood. This variety of sputum is, however, comparatively rare. The sputum is sometimes like that of bronchiectasis and sometimes has no particular features. It is not infrequently absent.

11. When a **tropical abscess** of the liver ruptures into the lung the sputum is reddish-brown and resembles anchovy sauce. The *Amœba histolytica* may be found in it.

12. In **pulmonary actinomycosis** the sputum is usually muco-purulent and may be fœtid. Sometimes it is mucoid, sometimes purulent, and may contain blood. The characteristic feature is the presence of "sulphur granules," yellowish, greenish, grey, or brown in

## STARVATION

colour, varying in size up to 2 mm. in diameter, and found to consist microscopically of fine, twisted threads terminating in the periphery as club-shaped swellings.

13. When a **hydatid cyst** of the lung bursts into the air-passages before suppuration has taken place, the sputum may be a clear, opalescent fluid containing a high sodium content. Daughter-cysts, scoleces, hooklets, or fragments of membrane may be found, each of which is pathognomonic. When suppuration has occurred the sputum becomes purulent and fœtid and may be chocolate-coloured. Gangrene of the lung may supervene and modify the sputum. Pieces of the cyst-wall may be expectorated for many weeks.

FREDERICK LANGMEAD.

**SQUINT** (*see* STRABISMUS).

**STAMMERING** (*see* SPEECH, DISTURBANCES OF).

**STAPHYLOMA** (*see* SCLERA, AFFECTIONS OF).

**STARVATION.**—Cases of starvation occur during famines, from accidental entombment in mines, etc., from wilful withdrawal of food, and from wilful refusal to take food. The majority of cases are the result of accident or homicide, but cases of wilful refusal to take food, sometimes ending in death, are met with among the fanatical and insane. While lay testimony will usually be sufficient to establish accident or suicide, medical evidence is important in trials for murder, as the defence usually put forward is that the inanition and death were due to disease and malassimilation. Whereas the deliberate withholding of food from a child so as to cause its death by starvation is homicide, the Children Act, 1908, Part II, sec. 12, declares that a parent or other person legally liable to maintain a child or young person shall be deemed to have neglected him in a manner likely to cause injury to his health if he fails to provide adequate food, clothing, medical aid, or lodging.

In healthy persons total deprivation of food and water will probably lead to a fatal termination in seven to ten days.

**Symptomatology.**—There is abdominal pain, relieved by pressure, and intolerable thirst; the lips are parched, the tongue is red and dry, the eyes are sunken, wild and glistening, with pupils dilated. There is progressive emaciation, bony prominences standing out, the

## STERILITY, FEMALE

extremities showing wasting and the abdomen being sunken. The voice is weak ("cholera voice"). The temperature descends to sub-normal; the pulse-rate is rapid at first, then slow, and again increases before death. The bowels are constipated, and any fæces passed are hard and ball-like; the urine is scanty and dark. The body gives off an offensive odour; the skin is wrinkled and shrivelled, petechiæ may be present, and a brown varnish-like material often covers the skin. Delirium and convulsions may precede death, but are often absent.

**Post-mortem examination.**—The external appearances are those already described. Internally there is disappearance of subcutaneous fat; the skeletal muscles are reduced in size, soft, and devoid of fat; the heart and kidneys are free from surrounding fat; and the liver, kidneys, spleen, and lungs are shrunken and comparatively bloodless. There is no fat in the omentum and mesentery, and the mesentery may have almost disappeared. The gall-bladder is full of dark bile, and the urinary bladder empty or nearly so. The stomach and intestines are contracted, collapsed, and so thinned that particles of food—and food may be given in homicidal cases shortly before death—may be recognized through their walls.

These appearances should be accompanied by an otherwise healthy state of the organs, since they may also be produced by organic disease. Since a defence of disease and inability to absorb and use the food that has been given will probably be set up, all appearances of disease should be noted carefully, especially conditions like tuberculosis, cirrhosis of kidneys, and glycosuria.

A. ALLISON.

**STATIC ELECTRICITY** (see ELECTRICAL TREATMENT).

**STATUS EPILEPTICUS** (see EPILEPSY).

**STATUS LYMPHATICUS** (see LYMPHATISM).

**STERILITY, FEMALE.**—This may be either absolute or relative. In the former condition a woman is unable to conceive; in the latter she is able to conceive but is unable to retain the fœtus until it is viable. A further variety is spoken of as "one-child sterility"; this is due to an acquired pelvic inflammation, usually of gonorrhœal origin, having its genesis during the puerperium of the first and only child, and resulting in such functional destruc-

tion of the internal genital organs that further conception is impossible.

**Causes.**—In a number of patients an adequate cause can be found, but quite often there are no physical signs. We may broadly classify the causes as follows:

### 1. Where no physical signs are found.

- (a) *Age.*—The most fertile women are those married between 20 and 25, of whom nine out of ten will conceive, while between 35 and 40 less than half will bear children. Too early marriage also is an unfavourable factor, only one in three bearing children.
- (b) *Sexual exhaustion* is a term used to denote a state produced by too frequent sexual intercourse. That it is a real cause is shown by the fact that most prostitutes are sterile.
- (c) *The ovaries* possibly may be *functionless* in respect of their power of ovulation.

### 2. Where physical signs are present.

- (a) *Defects of development* include imperforate hymen, imperfectly formed external genitals, rudimentary, infantile, and pubescent uterus, the conical cervix, and congenital hypertrophic elongation of the cervix.
- (b) *Vaginismus*, and *organic obstruction of the vaginal orifice* in the shape of a tough or cribriform hymen. Such conditions do not constitute an absolute bar to conception, for pregnancy has occurred without penetration.
- (c) *Fibroids* are often seen in sterile women, but it is doubtful whether they have been the cause of sterility, except where they are submucous or polypoidal and cause free bleeding. Subperitoneal fibroids are often seen in pregnancy.
- (d) *Pelvic inflammation* is only an absolute bar to fertility when the tubes are quite disorganized.
- (e) *Dyspareunia*, due to many causes.
- (f) *General disease*, such as advanced tuberculosis, obesity, myxœdema, alcoholism, and diabetes.

Conditions sometimes wrongly alleged as causes are endometritis, cervical lacerations and discharges, and sexual frigidity.

**Clinical examination.**—In any clinical examination of the female patient the foregoing points should especially be noted.

## STERILITY, FEMALE

Firstly, the age, height, and general appearance, together with the fertility of the families of both husband and wife; thus the tall thin and the obese types of women are often sterile. The vulva should be inspected to note the hymen, whether it is imperforate or cribriform. The ostium vaginæ may be unduly small, or vaginismus may be present. The cervix is frequently of a conical shape, so that the os is a very small orifice situated at its tip. Less often there is considerable elongation of the vaginal portion. The body of the uterus should be examined for its size, shape, position, degree of mobility, and the presence of fibroids. The size may be normal, but in sterile women it is often reduced even to the size of a mere nodule. All degrees between this rudimentary form and the normal are found, the commonest being the pubescent type, a globular-shaped fundus sharply anteflexed (less commonly retroflexed), and slightly smaller than the normal virgin uterus. It is frequently met with in spasmodic dysmenorrhœa, so often associated with sterility. A retroverted uterus, apart from incomplete development, has little influence on fertility except above the age of 30 (Herman). When the uterus is freely mobile, extensive pelvic adhesions are absent, but if the organ is fixed it is possible that the tubes have become occluded by inflammation. The tubes and ovaries are examined chiefly for evidence of inflammatory enlargement and fixation, and, rarely, double new growth of the ovaries. Congenital absence of the ovaries is not known and is never adduced as a cause of sterility. Prolapse of the ovary, as a cause of dyspareunia, should be noted.

**Treatment.**—There can be none for most of the congenital defects, the exception being abnormalities of the hymen. In cases of hymeneal obstruction the hymen should be excised. The conical cervix and anteflexed corpus uteri should be treated by wide dilatation of the cervix, associated in some cases with backward division of the vaginal portion, followed by the insertion of a uterine stem and a prolonged course of hot douching. Deep cervical lacerations should be repaired, and the congenitally elongated cervix requires amputation. The curette may profitably be employed for endometritis and polypi. Retroversion, if replacement is difficult, should be corrected by a pessary after replacement under an anæsthetic, or by Gilliam's operation. In some cases of salpingitis, where the abdominal ostium is closed, salpingostomy may be performed.

## STERILITY, MALE

Obesity is best treated by dieting and thyroid extract. In some cases where no cause can be found, the separation of husband and wife for a few weeks may be followed by conception.

A. W. BOURNE.

**STERILITY, MALE.**—Too often are childless marriages ascribed to some defect in the wife when it is the husband who is actually at fault. The relative proportions in which sterility occurs in the two sexes are uncertain, and published statistics on the point vary greatly, but it would appear that male sterility may be the cause of about 50 per cent. of childless unions. Gross places it as low as 17 per cent., Hubner found it to be 59 per cent., whilst Vedder regards male sterility as responsible for 70 per cent. The recognition of the frequency of sterility in the male is important, for not seldom have wives undergone surgical treatment which a careful examination of their husbands would have shown to be unnecessary.

On examination, the cause is obvious when gross abnormalities are found. Among them may be numbered congenital malformations and serious destruction of the testes or epididymes as by tuberculosis, syphilis, or malignant disease. Examination of the prostate and seminal vesicles should never be omitted, for sterility in a male in whom only one testicle or epididymis is diseased may be explained by involvement of these organs. According to Kenneth Walker, tuberculous epididymitis causes azoospermia in 85 per cent. of the cases, even though it is unilateral, chiefly because of an associated tuberculosis of the prostate. By far the commonest cause is antecedent gonorrhœa, for the detection of which careful examination of the prostate and of the posterior urethra may be necessary (*see GONORRHOEA*). Again, it is the incidence of chronic prostatitis and vesiculitis which determines the development of sterility in most cases.

Sometimes no malformation or signs of local disease are present. Here a careful history is of great importance, for it may indicate that impotence (*see SEXUAL FUNCTIONS, MALE, DISTURBANCES OF*) is the real cause of the failure to procreate. Alcoholism, exposure to X-rays, enfeebling diseases, sexual excess, and infections of the testicle and prostate other than those already mentioned, may lead to sterility, which, however, is usually temporary. Notable among the infections is mumps, which, if complicated by orchitis, especially in adolescents,

## STINGS AND BITES OF INSECTS

is likely to cause testicular fibrosis and atrophy.

Examination is incomplete until the male secretions have been investigated microscopically. Pus cells and organisms should be looked for, and the spermatozoa scrutinized in regard to their number and activity, and to the inclusion of degenerate forms. Such an examination may provide an explanation of barren unions when macroscopic evidence in the case both of husband and wife is lacking or inconclusive.

The treatment of sterility is that of its cause. For that resulting from impotence, see **SEXUAL FUNCTIONS, DISTURBANCES OF**.

FREDERICK LANGMEAD.

**STILL'S DISEASE** (see **RHEUMATOID ARTHRITIS**).

### STINGS AND BITES OF INSECTS.—

The lesions caused by the common animal parasites which infest the human body, such as the different varieties of pediculi and the acarus of scabies, are considered under their respective headings. In addition to these there are numerous insects which cause lesions by means of stings or bites or by crawling over or burrowing into the skin. In this category are included the common flea, the bed-bug, the harvest-bug (see **HARVEST-BUG RASH**), the wood tick, the bird mite, gnats and mosquitoes, several varieties of flies, bees, wasps, spiders, moths, ants, and caterpillars. Insect bites are, in general, characterized by a central hæmorrhagic point surrounded by a raised red papule or wheal and attended by severe itching. As would be expected, the uncovered parts of the body, and the ankles and legs, are the parts most often attacked. The common flea causes a small erythematous macule or wheal with a central hæmorrhagic puncture. Chloroform is sometimes used to kill the insect, a few drops being poured on to the clothing, and a weak carbolic or other antiseptic and antipruritic lotion will serve to allay the irritation. The bed-bug gives rise to a larger wheal with a central dark point and a considerable degree of surrounding hyperæmia. Antipruritic lotions such as lead lotion, weak solution of tar, or ichthyol give relief. Infested rooms and bedding must be properly disinfected. Gnats and mosquitoes and flies produce similar lesions, which are aggravated by scratching and are often accompanied by considerable oedema. Strong-smelling essential oils are useful as a prophylactic

## STOMACH, CANCER OF

measure, oil of lavender smeared on the exposed parts being one of the best. Oil of eucalyptus, citronella, and peppermint are also efficacious. Pyrethrum, naphthaline, or camphor in solution or ointment may be sprayed or rubbed on. The intense irritation and the swelling following the bites may be allayed by evaporating and antipruritic lotions, as mentioned above. Solutions of soda or ammonia are beneficial as antipruritics and to neutralize the acid injected by the insect. Thymol and menthol in ointment or solution are also valuable. A useful domestic remedy is a piece of washing soda rubbed on the affected part. Eau-de-cologne or vinegar is also serviceable as an antipruritic. If there is much swelling, compresses of lead lotion should be applied or a weak solution of ichthyol painted on. When the insect burrows into the skin, or leaves its sting in the puncture, it may be necessary to extract the offending body with a needle.

S. E. DORE.

**STOKES-ADAMS SYNDROME** (see **HEART-BLOCK**).

**STOMACH, CANCER OF.**—This is the only common form of new growth of the stomach. Benign growths such as polypi, fibromata, and lipomata have been described, but are rare and seldom cause serious symptoms. Gastric sarcoma also is extremely rare.

**Etiology.**—The stomach is very liable to cancer, and ranks next to the uterus in the incidence-rate of organs affected, and statistics show that its frequency is increasing. Males are more often affected than females, the ratio being 5 to 4. Eighty per cent. of cases occur between the ages of 40 and 70, and 10 per cent. between 20 and 40. There appears to be no definite relationship to previous dyspepsia, alcoholism, or trauma. It is said that cancer sometimes develops in a chronic gastric ulcer, especially if the latter is situated near the pylorus. In the opinion of most physicians this event is uncommon, but surgeons hold the view that the sequence is common. Statistics indicate that it occurs in from 5 to 10 per cent. of cases of gastric ulcer.

**Pathology.**—The pyloric region of the stomach is most commonly affected, next often the lesser curvature, and then the cardia; any part of the stomach, however, may be the seat of growth. Gastric cancer is usually primary, only about 1 per cent. of cases being secondary. The growth may be circumscribed,

## STOMACH, CANCER OF

forming perhaps a large fungating mass projecting into the stomach cavity, or may be diffuse and spread extensively in the wall of the organ. Two main types, the scirrhus and the medullary, are found; in the former much fibrous tissue exists with the epithelial cells. Medullary cancer is a cylindrical-celled epithelial growth in which colloid changes may occur.

If cancer involves the cardiac portion of the stomach the organ is usually reduced in size, but when the pylorus is the site, so that obstruction is caused, great gastric dilatation ensues.

In diffuse scirrhus cancer the stomach-wall is greatly thickened, and the cavity may be much contracted.

Secondary growths in other organs are of frequent occurrence, the lymphatic glands, liver, peritoneum, omentum, intestine, and pancreas being most often invaded. Sometimes the skin around the umbilicus or in the epigastric region is affected—a point of great value in diagnosis.

**Symptomatology.**—The symptoms may be latent, and death may occur in the absence of any indication of a gastric lesion. In other cases the symptoms may be those of progressive emaciation with anæmia resembling pernicious anæmia, and cachexia, there being nothing pointing to a disease of the stomach. Usually the early symptoms are anorexia, nausea, pain, dyspepsia, vomiting, loss of weight, and anæmia.

Progressive emaciation is one of the most constant features and may amount to a loss of several stones; in some cases, however, the loss in weight is not considerable. Progressive weakness is usually associated with the emaciation.

Pain is a common early symptom; it is usually epigastric, but may be referred to the interscapular or the infrascapular region, and is generally aggravated by the taking of food.

The anæmia is of the secondary type, and is often associated with a lemon-yellow tint of the skin. The hæmoglobin is much reduced, being frequently below 50 per cent.

If the cardiac orifice is involved, vomiting occurs almost immediately after meals; if the pyloric, a considerable time after—often in the evening—a large quantity of vomit being brought up. When neither of these orifices is affected, the vomiting recurs at frequent intervals after the taking of food.

The blood vomited is dark brown in colour,

and is unlike the bright-red or coffee-grounds vomit of gastric ulcer; it is not often profuse.

Constipation is usually present.

Abdominal inspection may show a fullness or projection in the epigastric region due to the underlying tumour, the movement with respiration being visible.

With pyloric cancer, peristalsis from left to right in the region of the stomach may be visible.

There may be felt in the stomach region a tumour which is movable on respiration and tender on pressure. Pyloric tumours vary in position with the degree of dilatation of the stomach, and may be either to right or to left of the middle line. Dullness is usually present over the tumour, and in the case of pyloric tumours percussion or auscultato-percussion reveals the extent of the gastric dilatation.

**Secondary growths** are common, and enlargement of the lymphatic glands of the neck or axillæ is frequently found. Involvement of the liver may be shown by its enlargement and tenderness. Other tumours may be felt in the abdomen, due to invasion of the omentum or peritoneum or of the intestine. Secondary growth in the intestine may cause symptoms of intestinal obstruction with dilatation of the bowel above the lesion. The pancreas, too, may be the site of secondary growth, and, if its head be affected, persistent jaundice is present, often accompanied by oedema of the feet from pressure on the inferior vena cava.

**Perforation.**—Not infrequently the growth in the stomach becomes adherent to the transverse colon and a perforation occurs between the two. The character of the vomit in such cases reveals the existence of the lesion, for it gives a marked reaction to stercobilin and usually presents some faecal characters on inspection. Perforation leading to acute general peritonitis is rare, but cancerous peritonitis due to invasion of the peritoneum with growth, and resulting ascites, is not uncommon.

**Diagnosis.** Examination of the gastric contents withdrawn one hour after a test meal usually reveals an entire absence of free hydrochloric acid. The active hydrochloric acid is much diminished, being usually well below 0.1 per cent. Mucin is generally present, and a pronounced lactic-acid reaction is common. The gastric ferments are absent or much reduced in amount.

X-ray examination after a bismuth carbonate or barium sulphate meal will usually provide evidence of growth, and the irregularity of

## STOMACH, CANCER OF

outline of the stomach and alteration of its normal movements will be shown. (See PLATE 46, Fig. 6, facing p. 557.) In cases where pyloric obstruction is the prominent feature there will be, in addition, the evidence of abnormal retention of the gastric contents.

Examination of the faeces for occult blood, the patient being kept on a milk diet and no meat or fish being allowed, will lead to the detection of blood in the stools.

**Prognosis.**—When a large tumour is present, or when there is evidence of secondary growths, the prospects of cure are hopeless.

When, however, the diagnosis is made in the early stage of the disease there is some prospect of recovery from operative treatment, but even in these cases the mortality is high and the prognosis grave.

**Treatment.**—If the diagnosis is established when the symptoms are of short duration, surgical treatment is indicated, for it offers the only hope of cure; removal of the growth in its entirety by gastrectomy more or less complete is necessary.

When the cardiac orifice is affected the insertion of radium tubes may relieve the obstruction; if this fails, gastrostomy and feeding through the artificial opening may be necessary. When the growth is at the pylorus and obstruction is present, should eradication be impossible, gastro-jejunostomy may be performed as a palliative measure.

Unfortunately, by the time a diagnosis is made, complete removal of the growth is usually impossible. In such cases the patient, having put his house in order, requires palliative treatment. Happily, the pain and discomfort are not usually extreme, and the symptoms can generally be kept under control. It is unwise to give morphine until absolutely necessary. Often phenacetin 10 gr., or pyranidon 6 gr., or a combination of these, will relieve the pain. A mixture such as the following helps digestion and gives relief; when pain is present, liq. morphinæ hydrochloridi 10 min. may be added.

R̄ Acid. hydrochl. dil. ℥xx.  
Glycer. pepsin. ʒi.  
Syr. aurant. ʒi.  
Tinct. cardam. co. ʒi.  
Aq. chlorof. ad ʒi.

Three times daily, half an hour after food.

During the later stages of the disease hypodermic injections of morphine should be employed as often as is necessary to relieve all pain.

W. H. WILLCOX.

## STOMACH, FUNCTIONAL DISORDERS

**STOMACH, DILATATION OF** (see STOMACH, FUNCTIONAL DISORDERS OF).

**STOMACH, EXAMINATION OF CONTENTS OF** (see GASTRIC CONTENTS, EXAMINATION OF).

**STOMACH, FUNCTIONAL DISORDERS OF.**—Omitting gastric flatulence, which is considered separately (see FLATULENCE), these affections may be divided into four groups:—

- I. MOTOR DISORDERS.
- II. SECRETORY DISORDERS.
- III. SENSORY DISORDERS.
- IV. GASTRIC SYMPTOMS OF FUNCTIONAL NERVOUS DISORDERS.

### I. MOTOR DISORDERS

#### 1. ATONY (ATONIC DILATATION)

**Etiology.**—Atony of the stomach and the dilatation to which it gives rise result most frequently from neurasthenia. It is also an occasional sequel of infections, especially influenza and typhoid fever, of anæmias, and of all conditions leading to malnutrition.

**Symptoms.**—The chief symptom is an uncomfortable sensation of fullness during and after meals, which leads to progressive diminution in the amount of food taken, the patient thereby hoping to diminish his discomfort, but actually increasing the atony by starving his nervous and muscular tissues. In uncomplicated cases pain and vomiting are never present.

**Diagnosis.**—The diagnosis of functional atonic dilatation depends firstly upon the estimation of the size and tone of the stomach, and secondly upon its differentiation from the atony and dilatation which occur in organic pyloric obstruction when the compensatory hypertrophy proves insufficient.

Although percussion gives some indication of the quantity of gas in the stomach, it does not help in the determination of the size of the viscus. Splashing and succussion occur in the normal stomach after an ordinary meal; if, however, they can be produced after 2 oz. of water have been sipped when the stomach is empty, atony is probably present. But the only method by which any definite information as to the size and tone of the stomach can be obtained without using the X-rays is that of inflation. The patient drinks on an empty stomach, in rapid succession, two quantities of water in which are dissolved respectively 1½ dr. of sodium bicarbonate and 1½ dr. of

## STOMACH, FUNCTIONAL DISORDERS OF

tartaric acid. At the body-temperature and atmospheric pressure, 1,700 c.c. of carbon dioxide are evolved. The normal stomach has a capacity of 600 to 1,200 c.c. when rapidly filled; 1,700 c.c. of gas are therefore subjected to a considerable degree of tension, and much discomfort is felt, which at once disappears on sitting up, when the gas is readily and forcibly expelled. If the tone of the stomach is deficient, its capacity even when empty is greater than 1,700 c.c.; consequently it is not tightly distended, and the tumour it forms is softer and its outline less easy to determine by palpation and percussion than normally. No discomfort is produced, and, on sitting up, the gas is expelled with difficulty. Normally the distance between the lesser and greater curvature should not be greater than 4 in., and the greater curvature should not reach below the umbilicus. Dilatation can thus be recognized and distinguished from mere ptosis without dilatation.

The size, shape, and position of the stomach can only be accurately determined by means of the X-rays (*see* PLATE 46, Figs. 1-4, facing p. 557) after the consumption of half a pint of porridge and milk containing 2 oz. of barium sulphate. Owing to the adaptation of the stomach to the volume of its contents, there is little difference in the upper level of the semi-fluid chyme as seen in the erect position, whether the volume is 5 oz. or 2 pints. In atonic dilatation the stomach no longer adapts itself accurately to the volume of its contents; food taken when it is empty drops at once to its most dependent part instead of being held up for a few seconds by the tonic contraction of the body of the stomach. As more and more food enters, the upper surface of the semi-fluid gastric contents gradually rises, but it never reaches the height which is attained under normal conditions following the entry of the first two or three mouthfuls into the stomach.

Peristalsis frequently remains normal in spite of the presence of a considerable degree of atony. Except in extreme cases, the gastric contents begin to pass into the duodenum immediately after their entry into the stomach, and the evacuation continues at the normal rate. Only the last traces of food remain abnormally long in the stomach, owing to the absence of the final increase of tone, which should cause the lower border of the stomach to rise above its original level just before it becomes completely empty, as the contents of

the most dependent part of the stomach are unable to pass into the pyloric vestibule. In pyloric obstruction the delay in the evacuation of the stomach is much greater. If the shadow of the stomach is still visible six hours after a barium meal, nothing having been taken in the interval, stasis is present; after nine hours it is probably, and after twelve hours it is certainly, due to organic pyloric obstruction.

Equally conclusive evidence can be obtained by means of the stomach-tube. A dinner, which should include meat and green vegetables, pudding and raisins, is given at 9 p.m. and the tube is passed at 9 a.m., nothing having been taken in the interval. If nothing can be removed by means of an "evacuator," half a pint of water is poured through the tube; when this is returned it will bring with it any traces of food the bulk of which was insufficient to be expelled alone. In atonic dilatation the stomach is always empty twelve hours after a meal; in organic obstruction more or less food is always present.

**Treatment.**—Rest in the recumbent position is not only valuable for the depressed condition of the nervous system, but it also places the atonic stomach in a condition of mechanical advantage, especially if the patient lies on his right side. In slight cases it is only necessary to lie down for an hour after each meal; in advanced cases the patient should at first remain in bed all day, and should lie on his right side while he eats and for an hour after each meal.

An ordinary mixed diet should be given, but the bulk of the meals should be as small as is compatible with the provision of a proper amount of nourishment, and fluids should be drunk only when the stomach is empty. A glass of milk may be taken on rising in the morning, a glass of milk an hour before lunch, one or two cups of tea at 5 p.m., and a glass of milk last thing at night. In severe cases no fluid at all should be drunk for some days, the meals being quite dry; two or three pints of normal saline solutions are administered per rectum either continuously or in two or three injections a day. Carbohydrates need not be restricted in uncomplicated cases, but when catarrh is present the quantity of sweet and starchy foods should be reduced. Abdominal massage is of great value.

Strychnine is often of use, as it improves the appetite, though it is doubtful whether it has any direct effect upon the gastric tone, and it



## STOMACH, FUNCTIONAL DISORDERS OF

should not be given to patients suffering from severe neurasthenia. It may be combined with dilute hydrochloric acid (15 min.), particularly if there is deficiency in gastric secretion, and with a bitter such as gentian, which stimulates the appetite and consequently increases the psychical secretion. In excitable patients, and in patients who find it difficult to lie quietly in bed for a prolonged period, small doses of bromide and arsenic should be given.

In uncomplicated cases, lavage is likely to do more harm than good, but in severe cases complicated by catarrh, lavage with small quantities of plain water may be of use. Gastro-enterostomy is absolutely contraindicated.

### 2. ACUTE DILATATION

**Etiology.**—After abdominal operations with or without peritonitis, and much less frequently in the course of acute and chronic infections, especially pneumonia, the stomach suddenly becomes greatly dilated owing to a complete loss of tone. The acute dilatation leads to obstruction of the duodenum by the mesentery at the point where the latter crosses it, and so the dilatation is greatly increased.

**Symptoms.**—The abdomen is much distended, large quantities of dark but not faecal fluid are vomited, and the patient rapidly becomes very collapsed.

**Treatment.**—The stomach should be emptied by an Einhorn tube kept continuously in position. Nothing should be given by mouth, but saline solution should be injected into the rectum or subcutaneously. The hips should be kept raised and the shoulders low. If improvement does not rapidly occur, a jejunostomy should be performed.

### 3. HYPERTONUS

**Etiology.**—When food is taken in small quantities at frequent intervals the stomach is never distended and its tone consequently increases: thus hypertonus is often seen in patients who have been strictly dieted for some weeks, especially if they have been kept in bed. The abnormally rapid evacuation of the stomach in achylia gastrica leads to the same result, well-marked hypertonus being generally present, except when the condition is due to cancer.

Hypertonus is, however, most commonly associated with hypersecretion: this combination is found in all cases of duodenal ulcer, except those in which obstruction has led to dilatation of the stomach.

**Symptoms.**—The only symptom which depends upon the hypertonus itself, as distinct from any abnormality of secretion or organic disease with which it may be associated, is due to the fact that a comparatively small quantity of food is sufficient to produce a rise in intragastric pressure, which results in a feeling of fullness, independent of the nature of the food, but varying in degree with its bulk and the rapidity with which it is eaten. The rapid evacuation of the stomach which commonly occurs when it is hypertonic gives rise to a feeling of emptiness and hunger about three hours after meals; immediate relief occurs when anything is eaten or drunk.

**Diagnosis.**—Hypertonus can only be diagnosed with certainty by means of the X-rays. In the erect position the stomach is diagonal or even horizontal instead of almost perpendicular as in normal subjects, and the pyloric vestibule, instead of the greater curvature of the body of the stomach, is its lowest part, being situated a considerable distance above the umbilicus. Peristalsis is either normal or exaggerated, and the pylorus tends to be relaxed, so that the stomach empties itself with unusual rapidity.

**Treatment.**—Apart from the treatment of any condition with which hypertonus may be associated, it is important that regular intermediate feeds should be taken in addition to the ordinary meals, in order that the stomach should not be empty for too long a period at a time. The food, which should be mechanically and chemically unirritating, must be thoroughly chewed and the teeth kept in good order. Belladonna is the only drug which has the effect of diminishing excessive tone, but a tablespoonful of olive oil taken before meals helps to prevent the too rapid evacuation of the stomach.

### 4. SPASM: SPASMODIC HOUR-GLASS STOMACH

Spasm of a narrow band of circular muscle-fibres is occasionally observed in the absence of organic disease. More often it is produced reflexly by a gastric or duodenal ulcer, chronic appendicitis, or gall-stones. An ulcer on the lesser curvature is the most common of the causes, the position of the spasm corresponding with that of the ulcer.

**Symptoms and diagnosis.**—The spasm in the centre of the stomach leads to the production of an hour-glass stomach. There are no characteristic symptoms, but the spasm is often the only objective gastric sign which is

## STOMACH, FUNCTIONAL DISORDERS OF

present in the dyspepsia of chronic appendicitis and gall-stones. It is important to be able to recognize the functional nature of the condition, as many needless operations for supposed organic hour-glass stomach have been performed as a result of misinterpreting the appearance seen with the X-rays. In organic hour-glass stomach the neck passes from the inner side of the most dependent part of the upper segment of the stomach, and not as in the functional condition from its lowest extremity. Moreover, a spasmodic stricture varies in degree during a single examination and at different examinations, and never gives rise to sufficient obstruction to cause stasis for six hours or increased peristalsis in the proximal segment. A spasmodic stricture differs from the orthostatic stricture often seen in gastroparesis by persisting when the patient lies down, and by its disappearance on vigorous massage and sometimes after a hypodermic injection of  $\frac{1}{160}$  gr. of atropine.

### 5. VOMITING

**Etiology.** (1) *Central vomiting.*—Vomiting is only rarely under the control of the will. But a person who has vomited a number of times owing to some central, reflex or toxic cause, may suggest to himself that certain circumstances will invariably cause him to vomit. The hysterical vomiting which results is an extremely common sequel of vomiting due to other causes.

Various emotions, especially those of disgust and fear, may result in vomiting, especially in individuals with an abnormally excitable nervous system.

Certain organic nervous diseases, such as cerebral tumour and meningitis, are frequently accompanied by vomiting, which may also occur in compression due to an injury and in concussion. This is due partly to increased intracranial pressure, and partly to direct irritation of higher parts of the brain from which impulses pass to the vomiting centre. In the case of cerebral tumour the relief of intracranial pressure by trephining may therefore cause the vomiting to cease.

(2) *Reflex vomiting.*—Reflex stimulation of the vomiting centre is the most common cause of vomiting. The most important source is the stomach itself, irritation of the mucous membrane by abnormal constituents of the gastric contents, whether introduced in the food or resulting from bacterial decomposition, being a frequent cause. Gassing was a very

common cause of vomiting in soldiers, some of the irritant gas being dissolved in saliva, which was secreted in excess and then swallowed. Vomiting is particularly likely to occur if the mucous membrane is inflamed, or if an ulcer or a growth is present. Over-distension with food, especially if it occurs rapidly, as when a big meal is bolted, or if it continues for an abnormally long period as a result of pyloric obstruction, has the same effect. In all these cases more or less relief to the local symptoms results from vomiting.

Painful stimulation of any afferent nerves, but particularly of those passing from abdominal viscera, such as occurs in the attacks of pain associated with disease of the stomach, in biliary and renal colic, in appendicitis and in intestinal obstruction and peritonitis, often causes vomiting. Tickling the fauces is a common method of inducing vomiting; vomiting may also be excited reflexly from the lungs in phthisis and from the heart when it is dilated, and the vomiting of migraine is probably in most cases a reflex from the eyes, owing to errors of refraction or of ocular muscle-balance, or from the alimentary canal.

Sea-sickness is caused by a reflex arising from abnormal stimulation of the semicircular canals. The vomiting in diseases of the ear, in which the semicircular canals are directly or indirectly involved, as in Menière's disease, is of similar origin. The vomiting of early pregnancy is probably reflex; when persistent it is probably always hysterical.

(3) *Toxic vomiting.*—Some emetics, such as apomorphine, cause vomiting by direct irritation of the vomiting centre, and this is also the cause of postanæsthetic vomiting. Other emetics, such as warm water containing salt, and copper and zinc sulphate, act reflexly from the stomach, and are consequently only effective when they are swallowed, whilst drugs such as tartar emetic and ipecacuanha act in both ways.

Poisons produced in the body, as in uræmia, Graves's disease and Addison's disease, may irritate the vomiting centre. In uræmia, however, the action is partly reflex, owing to the excretion into the stomach of toxins which should be excreted by the kidneys. The bacterial toxins in acute fevers, especially scarlet fever, often excite vomiting. Toxic vomiting differs from central vomiting and many cases of reflex vomiting in being almost invariably preceded by nausea.

**Treatment.**—The only treatment of vomit-

## STOMACH, FUNCTIONAL DISORDERS OF

ing is to remove the cause. In toxic cases, and those due to reflexes from the stomach itself, complete evacuation by the stomach-tube gives relief. As purely symptomatic treatment, the most useful drugs are dilute hydrocyanic acid, 3-6 min. of which should be given in a drachm of water, and chloretone, 5 gr. of which can be given in a cachet, and repeated, if necessary, three or four times at intervals of two hours. Chloretone is particularly useful for sea-sickness. It tends to produce drowsiness and may cause considerable malaise; it is therefore unsuitable for frequent administration. Where chloretone does not suit, sea-sickness may be controlled by taking 2 dr. of syrup of chloral and 30 gr. of potassium bromide in 1½ oz. of water in teaspoonful doses every five minutes until relief occurs or the patient falls asleep.

In severe cases of vomiting, especially when due to organic disease of the nervous system or painful diseases of other organs, morphine is required. Tincture of iodine, in doses of 2 min. taken every hour, 1-1½ gr. of cocaine hydrochloride, 5-10 gr. of cerium oxalate, and the salts of bismuth are often recommended, but I have never been able to convince myself that they have any effect.

### 6. REGURGITATION

Regurgitation of small quantities of food into the pharynx and less often into the mouth without effort and without nausea occurs in various forms of dyspepsia, especially when flatulence is present. It is often accompanied by a scalding sensation (pyrosis) to be described later (p. 269). It must be distinguished from regurgitation of saliva from the oesophagus—so-called waterbrash (p. 269). No special treatment is required beyond that of the primary condition; the momentary discomfort is relieved by drinking some water or taking a little soda or magnesia.

### 7. RUMINATION (MERYCISM)

**Etiology.**—Rumination may occur in several members of the same family. It occasionally develops as a result of imitation, and is most frequent in neurotic individuals, epileptics, and the insane. It generally begins between the ages of 12 and 20.

**Symptoms.**—Rumination consists in the return into the mouth of successive portions of a considerable part of the gastric contents after the completion of a meal. The food is chewed again and then swallowed; the pro-

cess is not at all distasteful and appears quite natural to the patient. This serves to distinguish rumination from regurgitation; moreover, it is never associated with dyspepsia.

**Treatment.**—In some cases suppression of rumination makes the patient feel unwell. When this is not the case, an attempt should be made to control it by an effort of will. Food should be eaten slowly, and fluids taken as much as possible apart from meals.

## II. SECRETORY DISORDERS

### 1. EXCESSIVE SECRETION OF GASTRIC JUICE (HYPERSECRETION, HYPERCHLORHYDRIA)

It was formerly believed that hyperchlorhydria, or the secretion of gastric juice containing an abnormally high percentage of hydrochloric acid, was the chief form of excessive secretory activity. The percentage of hydrochloric acid in the gastric juice cannot, however, be increased by any experimental means, although the quantity secreted can be readily increased. Normal undiluted human gastric juice contains between 0.35 and 0.55 per cent. of free hydrochloric acid, while the normal percentage of active hydrochloric acid (free and organically combined HCl) after a test-breakfast is from 0.1 to 0.2, and 0.55 is a greater percentage than that found in the severest case of "hyperchlorhydria." It is thus uncertain whether gastric juice containing an excessive percentage of hydrochloric acid is ever secreted and whether so-called hyperchlorhydria is not always a result of hypersecretion.

The secretion of an excessive quantity of gastric juice—hypersecretion—occurs (a) most frequently during digestion, (b) in paroxysms independently of digestion, and (c) continuously.

#### (a) DIGESTIVE HYPERSECRETION

**Etiology.**—In digestive hypersecretion an abnormally large amount of normal gastric juice is secreted in response to the stimulus of a meal. The volume of gastric contents removed after a test-breakfast is therefore excessive, and the percentage of active and of free hydrochloric acid is increased owing to the relatively high proportion of gastric juice compared with food in the gastric contents.

The amount of gastric juice secreted as a result of a given stimulus varies considerably in different individuals and in different nations, the chief determining factor being probably the diet, and some perfectly healthy people have

## STOMACH, FUNCTIONAL DISORDERS OF

what may be termed digestive hypersecretion, although they suffer from no gastric symptoms.

The commonest cause of slight hypersecretion is the chronic irritation caused by habitual over-eating and insufficient mastication, especially if much indigestible food, strong tea, or alcohol is consumed, or much tobacco is smoked. Gastric hypersecretion may therefore precede and accompany the early stages of chronic catarrhal gastritis.

The dyspepsia of chlorosis is often associated with hypersecretion and is probably due to irritation by the excess of tea and indigestible carbohydrate food which is often consumed by chlorotic girls.

Severe digestive hypersecretion is almost constant in duodenal ulcer; it is less constant and somewhat less marked in pre-pyloric ulcer, but is always present when an ulcer involves the pylorus. It does not occur at all in ulcers situated in other parts of the stomach. In duodenal ulcer the hypersecretion is probably primary, its association with hypertonus being the expression of the diathesis which makes an individual liable to the development of a duodenal ulcer. In pre-pyloric and pyloric ulcer the hypersecretion is probably secondary to the abnormal retention of food in the stomach.

**Symptoms.**—The chief symptom is *heartburn* or *pyrosis*. A burning sensation is felt in the epigastrium, which is often accompanied by a similar sensation behind the sternum and by the regurgitation into the pharynx of a small quantity of scalding fluid. The heartburn begins two or three hours after meals, and is relieved by drinking, eating, and by alkalis. A small quantity of very acid fluid, which generally contains fragments of vegetable food but no meat, may be vomited, complete relief resulting. In the absence of ulceration tenderness is slight and ill defined. The patient often complains of flatulence when the heartburn is at its height. The appetite is good, and constipation is commonly present. Occasionally reflex salivation occurs at the height of the attack: when excessive, this runs from the mouth, or it is swallowed and accumulates in the lower end of the œsophagus, whence it is ejected in considerable quantities as an alkaline, opalescent fluid, the condition being known as *waterbrash*. This accumulation is due to *achalasia*—or absence of the normal relaxation of the cardia which should occur when each peristaltic wave reaches the lower end of the œsophagus, this relaxation

being inhibited by the excessive acidity of the gastric contents.

**Diagnosis.**—The presence of hypersecretion can only be recognized with certainty by means of a test-breakfast, or the still more valuable fractional test-meal, in which a small quantity of the gastric contents is removed from the stomach and analysed every quarter of an hour throughout the whole period of digestion. The proportion of fluid to solid removed is excessive, and the proportion of active hydrochloric acid is greater than 0.2 per cent. Hypersecretion having been diagnosed, it is necessary to exclude gastric and duodenal ulcer, gall-stones, and chronic appendicitis before the condition can be regarded as functional.

**Treatment.**—Treatment should primarily be directed to the removal of the cause. In all cases the diet should contain plenty of fats, which inhibit the flow of gastric juice, and of proteins, which combine with much of the hydrochloric acid, the secretion of which they call forth, but as little carbohydrates as possible, as they stimulate secretion without combining with the acid. The most valuable articles of diet are therefore milk, cream, butter, cream cheese, eggs, fish, chicken, and tender meat. Meat extracts, raw and coarse vegetables, pips and skins of fruit, unripe fruit, pickles, vinegar, mustard, pepper, curry, cheese (except cream cheese), new bread, alcohol, and effervescing drinks must be avoided. The food must be slowly eaten and thoroughly chewed. As symptomatic treatment, the patient, at the first suspicion of discomfort, should take a very small quantity of a powder containing equal parts of magnesia, calcium bicarbonate, and bismuth oxycarbonate, and should continue taking small doses until the pain is controlled.

### (b) PAROXYSMAL HYPERSECRETION

Paroxysmal secretion of an excessive quantity of gastric juice for a few hours or for several days, independently of the presence of food in the stomach, constitutes one form of the gastric crises of tabes, and may occur in association with headache in some cases of migraine (*see* p. 275).

### (c) CONTINUOUS HYPERSECRETION (REICHMANN'S DISEASE)

When a gastric or duodenal ulcer involves the pyloric canal the secretion of gastric juice becomes continuous; meals cause an increase in the flow, but it does not cease even when every trace of food has left the stomach. In

## STOMACH, FUNCTIONAL DISORDERS OF

such cases 50–1,000 c.c. of pure gastric juice are found in the stomach before breakfast. During meals the gastric juice is diluted, and most of the hydrochloric acid combines with the protein of the food, but in the intervals there is nothing but the gastric mucus and the mucus and sodium bicarbonate of the saliva with which it can combine. Consequently a higher percentage of free hydrochloric acid and of active hydrochloric acid is present in the intervals between meals than immediately after meals, although the strength of gastric juice secreted probably remains constant. A test-breakfast or fractional test-meal gives the same result as with digestive hypersecretion, but instead of the stomach being empty before breakfast it contains a quantity of very acid fluid, food being absent unless the condition has become complicated by definite obstruction.

The symptoms and treatment are those of pyloric ulcer; recent observations have shown that if care be taken to neutralize with alkalis every trace of acid secreted throughout the day and to keep the stomach empty during the night by means of a stomach-tube, the ulcer will often heal without leading to pyloric obstruction.

### 2. DEFICIENT SECRETION OF GASTRIC JUICE (HYPOSECRETION, HYPOCHLORHYDRIA, ACHYLIA)

The secretory activity of the stomach may be regarded as deficient when free hydrochloric acid is absent and the percentage of active acid is less than the normal minimum of 0.1 per cent. It may manifest itself in the secretion of an abnormally small quantity of normal gastric juice—*hyposecretion*, or of juice containing an abnormally small percentage of hydrochloric acid—*hypochlorhydria*. In both conditions there is, as a rule, a corresponding diminution in the quantity of ferment. As it is usually impossible to distinguish between hyposecretion and hypochlorhydria with certainty, they will be discussed together. When the quantity of hydrochloric acid is very deficient or none at all is secreted, the condition is called *achylia gastrica*. Even in the complete absence of hydrochloric acid, traces of the gastric ferments are almost invariably still present, but they are inactive unless acid is added to them.

**Etiology.**—In the not uncommon condition known as *primary achylia gastrica*, little or no gastric juice is secreted, probably as a result

of some congenital abnormality of the gastric mucous membrane.

**Secondary achylia gastrica.**—More often hyposecretion and hypochlorhydria are secondary to (1) some functional condition or (2) some organic disease of the stomach.

(1) The quantity of gastric juice secreted diminishes in the later years of life; free hydrochloric acid is absent from the gastric contents after a test-breakfast in more than 30 per cent. of all subjects over the age of 50.

Both hyposecretion and hypochlorhydria occur in acute fevers and all diseases which result in malnutrition. The secretion of gastric juice depends largely upon the stimulating influence of a healthy appetite and the pleasure derived from the sight, smell, and taste of a good meal. When, therefore, food is eaten quickly with a preoccupied mind, when it is distasteful or served under unpleasant conditions, or when the tongue is furred and the sense of taste impaired, the psychical stimulus is deficient, and the initial secretion of “appetite juice” does not occur. The hyposecretion, which is often present in neurasthenia, in phthisis, and other chronic infections, and after typhoid fever, is mainly due to these causes. In rare cases the complete inhibition of secretion which is produced by fear and other depressing emotions remains after the cause has apparently disappeared, but in all probability the mental disturbance is still present though in a less obvious form.

(2) Hyposecretion always occurs in acute gastritis. In the early stages of chronic gastritis, hypersecretion is present, but if the cause is not removed the quantity of hydrochloric acid sooner or later becomes subnormal. The amount of free hydrochloric acid is further reduced by combining with the mucus, which is always present in excess. In severe cases of acute gastritis and in severe and prolonged cases of chronic gastritis the gastric mucous membrane may become partially atrophied. This leads to hypochlorhydria in addition to the hyposecretion. The *achylia gastrica* which is constantly present in Addison's (pernicious) anæmia is possibly of this nature, but more probably it is of the primary variety and precedes the development of the anæmia, of which it is an essential predisposing factor.

Cancer of the stomach always results in a diminution and finally in the disappearance of free hydrochloric acid, and the percentage of active hydrochloric acid also becomes progressively smaller. These changes are in part

## STOMACH, FUNCTIONAL DISORDERS OF

due to the loss of the psychical secretion, as more or less complete anorexia is generally present, and to the chronic gastritis which is often present at the same time. There must, however, be some other and more specific cause, the nature of which is still obscure.

**Symptoms and diagnosis.**—When the deficient secretion of gastric juice is *secondary*, the symptoms to which it gives rise are to a great extent obscured by those of the primary condition. No doubt some of the local symptoms of gastritis and cancer of the stomach are a result of the hyposecretion which is present, as the administration of hydrochloric acid often improves the digestion in such cases. The same is true with regard to the indigestion of neurasthenia and those cases of senile dyspepsia in which hyposecretion is found. The part taken by hyposecretion in the production of the symptoms of gastritis and cancer and of neurasthenic dyspepsia can best be judged from a consideration of primary achylia gastrica, all the symptoms of which are due to the deficient secretion of gastric juice.

*Primary* achylia gastrica may be completely latent and discovered accidentally. More frequently there is a history dating from childhood of repeated attacks of abdominal discomfort with anorexia and nausea. In adult life these attacks tend to become more frequent and severe. They are generally brought on by a hurried meal or by an unusually large or indigestible one. In the intervals between attacks the patient is often not completely comfortable, and there is sometimes a permanent distaste for meat, which also tends to upset the patient more than any other food. The discomfort comes on immediately after meals and lasts for an hour or two, the duration depending upon the amount of food taken. Though nausea is common, there is generally no vomiting.

In many cases chronic diarrhoea is present, either alone or associated with the gastric symptoms just described (gastrogenous diarrhoea).

Not infrequently achylia gastrica is associated with chronic appendicitis. Improvement in the symptoms occurs both on giving hydrochloric acid and after appendicectomy, but recovery is incomplete unless the two treatments are combined. Appendicectomy does not lead to the reappearance of gastric secretion, as the achylia is primary and the appendicitis caused by secondary infection.

In uncomplicated cases there is no tenderness,

and nothing abnormal is found on physical examination. The X-rays often show that the stomach is very small and hypertonic, and empties itself with extreme rapidity owing to the relaxed condition of the pyloric sphincter, which offers an abnormally slight resistance to the normal peristalsis. Owing to this the patient may complain of a sinking feeling about three hours after meals, which is at once relieved by taking more food.

Owing to the rapid evacuation of the stomach, it is often impossible to obtain any gastric contents when the tube is passed an hour after a test-breakfast. If this should occur, the possibility of achylia gastrica should at once be considered, and the tube should be passed three-quarters of an hour after a test-breakfast given the next morning. The gastric contents are so thick that they often cannot be removed without suction by some such apparatus as Senoran's evacuator. They are small in bulk, viscid, neutral or alkaline in reaction, and contain little or no active hydrochloric acid.

When the presence of achylia gastrica has been proved by means of a test-breakfast, it is necessary next to ascertain whether it is primary, or secondary to some other condition; the chief conditions which can be associated with gastric secretion so deficient that the term achylia can be correctly applied to it, are chronic atrophic gastritis, Addison's anaemia, and cancer of the stomach. The diagnosis is difficult owing to primary achylia gastrica and the achylia associated with Addison's anaemia being frequently complicated by chronic gastritis.

The existence of chronic gastritis is at once recognized by the presence of mucus in the gastric contents. The blood should always be examined; the presence of deficient red corpuscles and haemoglobin with a colour-index over 1.0, and the abnormal types of red corpuscles characteristic of Addison's anaemia, makes the diagnosis of this cause of achylia gastrica easy; secondary anaemia with a colour-index below 1.0 and normal red corpuscles suggests the possibility of cancer, as the blood is in every way normal in primary achylia. If the gastric contents contain blood, either obvious to the naked eye or only discovered by chemical means, cancer is probably present; occult blood should be looked for in the stools, as it is often found in cancer of the stomach even when it is absent from the gastric contents.

## STOMACH, FUNCTIONAL DISORDERS OF

If the X-rays show that the stomach is hypertonic and empties itself with abnormal rapidity, either primary achylia or the leather-bottle type of gastric carcinoma is present; in the latter the outline of the stomach may be irregular, and peristalsis is weak or even absent, the rapid evacuation being due to pyloric incompetence from a rigid and dilated sphincter. In achylia from gastritis the stomach is normal in size or dilated, whereas if the gastritis is secondary the appearance characteristic of primary achylia is seen. In all cases of cancer except the leather-bottle type, the stomach is normal in size or dilated, evacuation is often delayed, and in most cases some irregularity in the outline can be recognized.

**Treatment.**—The food should be chewed until it is quite fluid. All raw vegetables, such as salads and pickles, and other food which cannot be chewed to a fluid consistence, must be avoided. Very little meat should be eaten, and neither game nor ripe cheese allowed. If chronic gastritis, whether primary or secondary to the achylia, is present, the diet should at first be much more strict (see GASTRITIS, CHRONIC).

The only drug of real value is hydrochloric acid, but if hyposecretion and not complete achylia is present, a bitter mixture containing sodium bicarbonate taken before meals may stimulate secretion. Large quantities of hydrochloric acid should be given, and the treatment continued for the rest of the patient's life in all cases of primary achylia and of achylia secondary to gastritis (after the latter is cured), as well as in the achylia of Addison's anæmia, in which it not only improves the digestion but exerts a favourable influence on the disease itself. Small quantities of acid are required as symptomatic treatment for the hypochlorhydria or achylia of neurasthenia, acute and chronic infections, chronic gastritis and cancer. In chronic gastritis the secretion of gastric juice sometimes returns when the condition improves; a test-meal should therefore be given from time to time to see whether it is necessary to continue the administration of the acid.

Fifteen minims of dilute hydrochloric acid should be taken once or twice during each meal according to its size, at the end of the meal, and again a quarter of an hour and half an hour afterwards. Each dose may be taken in an ounce of water, or all the doses except those taken a quarter and half an hour after the meal may be given together mixed with the

water which the patient drinks during his meal. If some egg-albumen is added in both cases the liability of the mouth to become sore disappears; the further addition of sugar with or without lemon juice makes the drink quite palatable, and most patients eventually become accustomed to it. Some pepsin may also be taken, but in most cases it is not really required, as its secretion is rarely diminished to an extent likely to impair digestion.

### III. SENSORY DISORDERS

#### 1. EXCESSIVE APPETITE (BULIMIA)

Bulimia may result from the necessity of compensating for excessive metabolism in diabetes and Graves's disease and for the loss of nutrient material in chronic diarrhoea. It also occurs with pregnancy and intestinal worms, and it may be hysterical. In a slight form it is present in conditions, such as duodenal ulcer, which are associated with gastric hypermotility and hyperacidity.

**Treatment.**—The treatment of the primary condition is the chief indication. When bulimia is leading to obesity, as it frequently does after the age of 50, a mixture containing bromide of potassium 5 gr., with tincture of belladonna 5 min., taken half an hour before meals, may diminish the appetite. Where this has failed, I have found tincture of opium 7 min. effective. In such cases it is advisable to appease the appetite by means of food which is bulky but not nutritious, such as clear soups, green vegetables, and salads.

#### 2. LOSS OF APPETITE (ANOREXIA)

Anorexia occurs in gastritis and cancer of the stomach and in toxæmic conditions, such as acute fevers, tuberculosis, and cancer. It is often present when for any reason the tongue is dry or furred. It is an extremely common symptom in neurasthenia. Neurotic girls sometimes take too little to eat for fear of becoming fat. In all such cases the diminution in the quantity of food eaten leads to a corresponding diminution in the appetite, until in severe cases the condition known as *anorexia nervosa* finally develops, the patient taking little or no food and becoming excessively emaciated. (See NEURASTHENIA.)

**Treatment.**—The anorexia of neurasthenic and hysterical patients can only be overcome by persuading the patient to eat more. This is often very difficult and requires considerable tact. A strong-willed nurse is a great help,

## STOMACH, FUNCTIONAL DISORDERS OF

and food should be given at short intervals throughout the day. As soon as the patient begins to eat an increased quantity, the appetite returns. In severe cases complete isolation is essential, but with vigorous psychotherapy feeding through a tube should never be necessary, even in the worst cases of hysterical anorexia.

### 3. ABNORMAL SENSATIONS ACCOMPANYING HUNGER

In neurasthenic and hysterical individuals a distressing sense of hunger may suddenly appear, especially when the patient knows that no food is at hand; it is often associated with a feeling of great anxiety and exhaustion, and sometimes with cold sweats, vertigo, and nausea. In neurasthenia a few mouthfuls are sufficient to make the patient comfortable, but in hysteria the condition may be associated with bulimia. I have observed a similar sensation replace the hunger-pain in duodenal ulcer, and also in a case of achylia gastrica.

**Treatment.**—In addition to the general treatment for neurasthenia and hysteria the patient should always carry a little food in his pocket and have some by his bedside, so that he can take it the moment he feels hungry; this prevents the development of the other symptoms.

### 4. DEPRAVED APPETITE (PAROREXIA)

Parorexia may occur in hysterical and insane patients and in a mild form in chlorosis; the patient has a longing for very acid or highly piced food, and the insane may crave for non-edible articles, such as earth. (*See also PICA.*)

### 5. ABSENCE OF SATIETY (AKORIA)

In akoria the patient always feels empty, even after a large meal. In uncomplicated cases there is no craving for more food, such as occurs with bulimia, but the two conditions may be associated. Akoria is an important factor in the production of obesity in some individuals, and especially if associated with bulimia.

**Treatment.**—The patient should be instructed to take only a defined amount of food, even if he does not feel satisfied with it, and he should select bulky, non-nutritious articles of diet. Small doses of bromide and belladonna are of service if bulimia is also present, but otherwise they are not required.

### 6. HYPERÆSTHESIA

The excessive irritability of the central nervous system in neurasthenia and chlorosis results in a lowering of the minimal adequate stimulus and an exaggeration of the sensory response to visceral stimuli. Consequently digestive processes, which are normally imperceptible, become perceptible, and conditions which would otherwise lead to nothing more than a sensation of slight discomfort give rise to pain. Neurasthenic subjects tend, moreover, to be hypochondriacal and to concentrate their attention on every slight sensation in their digestive organs, thereby further increasing visceral sensibility. Gastric hyperæsthesia also results from increased irritability of the segments of the spinal cord, which receive the afferent fibres from the stomach, when other parts innervated by the same segments are the seat of disease. This accounts for the gastric hyperæsthesia associated with disorders of the biliary passages, appendicitis, and dilated heart. The hyperæsthesia may remain for some time after the primary condition is cured, as for instance after appendicectomy in cases of appendicular dyspepsia.

### 7. RAPID SATIETY

This is common in neurasthenia, the patient complaining of a sensation of fullness and distension as soon as he has taken a very small quantity of food, although he may immediately before have had a ravenous appetite. It is one cause of the inanition common in neurasthenia.

**Treatment.**—The patient should be persuaded to eat a normal amount of food in spite of the feeling of fullness, by impressing upon him that in order to get well he must go through a certain amount of discomfort. The sensation of fullness gradually disappears as his weight increases and his general condition improves.

For gastralgia, *see* p. 275.

### 8. NAUSEA

**Etiology.**—Nausea most commonly precedes vomiting, and is relieved when the stomach is emptied. It may, however, occur as an independent symptom, particularly in women. The majority of cases occur in chlorotic girls or at the climacteric. Although the significance of the symptom is not clear, it must be regarded in these cases as of nervous origin; probably it is often simply due to



## STOMACH, FUNCTIONAL DISORDERS OF

the perpetuation by auto-suggestion of nausea which originally resulted from the emotion of disgust, although the actual exciting incident may have been forgotten.

**Symptoms.**—The nausea may be constant, or periodic; it is often quite independent of the kind or nature of the food taken. The patient may wake up with severe nausea, which makes it difficult for her to take any food during the day. Moreover, she often thinks that the symptom is due to stomach disease; she consequently reduces the amount of food she takes, and severe loss of weight may result. The nausea is often increased by emotional disturbances, and may be less troublesome when the mind is fully occupied.

**Diagnosis.**—Nausea may be the only symptom in achylia gastrica, chronic gastritis, or even in the earliest stage of carcinoma of the stomach; it may be the chief complaint in chronic appendicitis and infection of the intestines with worms. It is occasionally a prominent symptom in early tuberculosis and in the commencement of pregnancy. All these possibilities should be excluded before regarding it as of nervous origin. The urine should be carefully examined, as nausea is occasionally an early symptom of nephritis and of infection of the urinary tract, and in men it may result from prostatic disease.

**Treatment.**—If all organic causes can be excluded, the only effective treatment is to remove the patient from her home surroundings and insist upon a proper quantity of food being taken. An attempt should be made to find the emotional disturbance which may have been the exciting cause of the symptom, as a convincing explanation of this is likely to lead to the disappearance of the nausea.

### IV. GASTRIC SYMPTOMS OF FUNCTIONAL NERVOUS DISORDERS

#### 1. NEURASTHENIC DYSPEPSIA

**Etiology.**—Most neurasthenic patients suffer from indigestion, partly as a result of gastric hyperæsthesia (p. 273) and partly as a result of depressing emotions, mental and physical overwork, and the other factors concerned in the production of neurasthenia, as these tend in themselves to inhibit the motor and secretory functions of the stomach. (See NEURASTHENIA.)

**Symptoms.**—The gastric symptoms in neurasthenia are characterized by their extreme irregularity, the patient feeling very ill one

day and quite well the next without any obvious reason for the change. The most constant complaint is of vague abdominal discomfort, which rarely amounts to actual pain. It is generally worst in the morning and improves towards evening, but in some cases the symptoms increase when the patient becomes more fatigued towards evening. The discomfort is worse after meals, a sensation of fullness being felt as soon as a small quantity of food has been eaten, but it may be present to a less extent before breakfast. It has little relation to the amount or the kind of food, differing in that way from the discomfort of organic gastric disorders; it is increased by worry or excitement, while some new interest, whether it be a change of surroundings, the visit of a friend, or a new medicine, leads to its temporary disappearance. Nausea sometimes occurs, but vomiting is unusual. Most patients complain of flatulence, which is more commonly due to aerophagy than to excessive fermentation, but may be nothing more than the result of misinterpreting the sensation of fullness, no excess of gas being present (see FLATULENCE). The appetite is always diminished, though it varies considerably from day to day. As insufficient food is taken, the nervous system becomes further depressed; this reacts again on the digestion, and so a vicious circle is produced.

Examination of the stomach shows that in some cases atonic dilatation (p. 264) is present; the secretion of gastric juice varies considerably from day to day, deficiency being rather more common than excess. Constipation is almost constantly present (see NEURASTHENIA). The abdominal muscles are often very tense, so that examination may be difficult, but there is never any great degree of tenderness, and what is present is diffuse and variable in position rather than localized and constant. The gastric symptoms are always associated with other symptoms of neurasthenia (q.v.).

**Treatment.**—The patient's confidence can only be gained after a very thorough examination. He can then be told that he has no organic disease, and that with perseverance he will get well. The general neurasthenic condition first requires attention; for this, mental and physical rest and sufficient food to overcome the inanition which is generally present are the chief indications. Pleasant surroundings, cheerful companions, and appetizing food are of great importance, and consequently

## STOMACH, FUNCTIONAL DISORDERS OF

strict isolation is rarely required. The treatment of the stomach itself depends upon the condition of the motor and secretory functions, and has already been described.

### 2. HYSTERIA

When symptoms such as vomiting and abdominal pain have been caused by gastritis, gastric ulcer, chlorotic dyspepsia or appendicitis, they may continue or recur as the result of suggestion after the original cause has disappeared. Many cases of persistent vomiting in pregnancy are really hysterical.

**Symptoms.**—*Hysterical vomiting* occurs during or immediately after meals; it is effortless and generally unaccompanied by nausea, so that a patient may vomit the meat and vegetables and immediately afterwards be ready to eat the sweets, which she may manage to retain. The stomach is generally only partially emptied, so that nutrition is preserved, but in rare cases nothing is retained and severe emaciation results. The diet has often no effect, vomiting being as frequent with milk alone as with a full diet.

*Gastralgia* is a not uncommon symptom in hysterical girls who have previously suffered from genuine gastric pain as a result of chlorosis or gastric ulcer. It is very irregular in its relations to the time and character of meals; it may occur before as well as after breakfast, and may be absent after a very indigestible meal during which the patient has had her mind distracted. It is accompanied by very diffuse deep tenderness, which may be associated with a large area of cutaneous hyperæsthesia, probably always produced by suggestion on the part of the observer, as its situation and extent can easily be varied from day to day by suggestion. The gastralgia may be associated with vomiting, which does not always relieve it.

**Treatment.**—In *hysterical vomiting* the patient should be kept in bed and isolated. It should be explained to her that the original cause of her vomiting has disappeared, and that her stomach has simply got into a bad habit, which it must be educated to give up. She should be given an additional helping of any food she vomits, even if this has to be repeated two or three times. In *hysterical gastralgia*, rest in bed, isolation, full diet and the application of a blister to the epigastrium generally effect a rapid cure, after the psychological factors concerned in its production have been ascertained and the nature of the condition

has been satisfactorily explained to the patient. (See also Aerophagy, under FLATULENCE, and Anorexia, p. 272.)

### 3. MIGRAINE

**Etiology.**—Whenever indigestion is accompanied by headache the possibility of migraine should be considered. In all cases an abnormal condition of the central nervous system is probably the essential factor. In most cases an error of refraction, particularly astigmatism, is present, often with some disturbance of muscle-balance, but when abdominal symptoms are well marked there is often some accessory exciting cause in the abdomen itself, the most common being constipation. As a nervous disease, migraine forms the subject of a separate article; here we are concerned more particularly with its abdominal manifestations.

**Symptoms.**—In typical cases an attack begins with ocular phenomena, which are followed by a unilateral headache, the abdominal symptoms only beginning when the headache has reached a considerable degree of severity. The most common symptom is a vague sensation of discomfort in the upper part of the abdomen, which rarely amounts to actual pain. Complete anorexia is present, and nausea is very common. In many cases vomiting finally occurs; this often results in the disappearance of the headache, and some patients for this reason make themselves vomit. Excessive secretion of gastric juice may take place during the attack, whether food is present in the stomach or not, and a certain amount of relief follows its neutralization by alkalis; in other cases there is achylia, and relief follows the administration of hydrochloric acid. In both cases the relief is mainly to the abdominal symptoms, though occasionally the headache improves also. Pressure on the abdomen generally increases the discomfort, but there is neither localized tenderness nor muscular rigidity.

In many cases the regular sequence of ocular phenomena, headache, abdominal discomfort, and vomiting is not observed, the only essential phenomena being recurrent attacks of abdominal discomfort associated with headache. Occasionally the headache may disappear completely, so that the nature of the recurrent abdominal attacks can only be recognized from the past history. The attacks usually begin on waking in the morning and often last all day. They may be extremely frequent, or only recur once or twice a year

## STOMACH, FUNCTIONAL DISORDERS

**Treatment.**—The general treatment is described in the article **MIGRAINE**. Drugs which relieve the headache do not greatly influence the gastric symptoms, which are best relieved by alkalis, especially sodium bicarbonate, by warm applications, and in severe cases by vomiting, induced by tickling the throat or by an emetic, or evacuation of the stomach by a tube at the first sign of an attack.

### 4. GASTRIC CRISES OF TABES

Gastric crises generally occur in the pre-ataxic period of tabes and often disappear as the more obvious symptoms develop.

**Symptoms.**—The symptoms appear and disappear with extreme suddenness, the patient being completely free from digestive symptoms in the intervals. The attacks may last from a few hours to several days; short attacks may occur daily, or the intervals may be as long as six months.

Pain and vomiting generally occur together, but either may be present alone. The pain is in the epigastrium and is excessively severe; it has no relation to meals and is only slightly relieved by vomiting. There is generally no tenderness. The vomiting is very intractable; the food last taken is first brought up, after which pure gastric juice (paroxysmal hypersecretion) or mucous fluid, often bile-stained and sometimes streaked with blood, is vomited. The pain and the vomiting lead to profound collapse.

**Diagnosis.**—Attacks of severe pain or vomiting, beginning and ending suddenly, uninfluenced by treatment, and of a character which does not correspond with that of any ordinary gastric disease, should arouse a suspicion of tabes. Even when there are no other symptoms of tabes, the knee-jerks, and still more frequently the ankle-jerks, are absent, the pupils may be abnormal, the cerebro-spinal fluid contains lymphocytes and the Wassermann reaction of the serum and cerebro-spinal fluid is generally positive.

**Treatment.**—The patient should take nothing but fluids, and when vomiting is persistent a continuous rectal infusion of normal saline or dextrose solution should be given. When there is hypersecretion, frequently-repeated drachm doses of sodium bicarbonate may relieve the pain. In other cases the pain is associated with a great rise in blood-pressure, and inhalation of amyl nitrite gives relief, but frequently nothing but morphia is effective.

A. F. HURST.

## STOMATITIS AND GLOSSITIS

**STOMACH, INFLAMMATION OF** (*see* **GASTRITIS**).

**STOMACH, MALPOSITION OF** (*see* **VISCEROPTOSIS**).

**STOMACH, RUPTURE OF** (*see* **ABDOMINAL INJURIES**).

**STOMACH, ULCER OF** (*see* **GASTRIC AND DUODENAL ULCERATION**).

**STOMATITIS AND GLOSSITIS.**—In this article are included—

1. EXUDATES AND DEPOSITS.
  - (a) Diphtheritic.
  - (b) Acute fibrinous.
  - (c) Thrush.
2. ACUTE SIMPLE STOMATITIS AND GLOSSITIS.
3. ULCERATION.
  - (a) Simple.
  - (b) Aphthous.
  - (c) Bednar's aphthæ.
4. STREPTOCOCCAL STOMATITIS.
5. GONORRHOEAL STOMATITIS.
6. MERCURIAL STOMATITIS.
7. GANGRENOUS STOMATITIS (**CANCERUM ORIS**).
8. SYPHILIS OF THE MOUTH.
9. AFFECTIONS OF THE GUMS.
  - (a) Hypertrophy.
  - (b) Spongy gums.
10. DISORDERS OF THE TONGUE.
  - (a) Fur.
  - (b) Black tongue.
  - (c) Geographical tongue.
  - (d) Lichen.
  - (e) Ulceration.

The reader is also referred to **PYORRHOEA ALVEOLARIS**; **EPULIS**; **TONGUE, NEW GROWTHS OF**; **TONGUE, SYPHILIS OF**; **TONGUE, TUBERCULOSIS OF**; and **TONGUE, ULCERATION OF**, under their respective titles.

Stomatitis may occur in the cheek, palate, gums, or tongue. Those affections which are localized principally or solely in the gums or tongue are dealt with separately, but in the great majority of cases of stomatitis all parts of the mouth share to some extent at least in the morbid process.

1. **Exudates and deposits** upon the mucous membrane of the mouth include (a) those of diphtheria, (b) acute fibrinous exudates, and (c) thrush.

(a) The characteristic features of buccal **diphtheria** are the greyish colour of the membrane, its bilateral distribution, its situation at the back of the mouth spreading on to the anterior pillars of the fauces and under-surface of the soft palate, the bleeding surface which is left on its removal, the general symptoms of

## STOMATITIS AND GLOSSITIS

diphtheria, and, lastly, the finding of the Klebs-Löffler bacillus. Generalized buccal diphtheria is a clinical rarity.

(b) **Acute fibrinous exudates** occasionally occur in severe and acute infection of the mouth. They resemble in many particulars the membranous septic exudates seen more often in the pharynx. Their distinction from diphtheritic exudates rests upon the relative rarity of the latter in the mouth, the higher temperature and greater apparent severity of the former, and the result of bacteriological investigation. Membranous stomatitis is usually streptococcal, and the outlook is serious; active local antiseptic measures are called for in combination with free use of iron, arsenic, and brandy. The value of antistreptococcic serum is problematical.

(c) **Thrush** is not truly a stomatitis; it is a condition in which a white mould develops in irregular patches upon the surface of the mucous membrane, which, except for a local redness, is itself normal. The fungus is the *Oidium albicans* which gains access to the mouth in contaminated food, especially milk. If the milk, feeding-vessels and mouth be kept thoroughly clean, thrush can obtain no foothold, but in poorly nourished, insufficiently tended infants the affection is very common. It is seen also, occasionally, in old cachectic patients who are nourished mainly upon milk. It appears on the tongue as white spots, and may afterwards spread to the gums, lips, buccal mucosa and palate. By coalescence the spots become merged to form greyish-white plaques. Occasionally it affects the pharynx, tonsils, œsophagus, stomach, or cæcum, and as a rare event the vocal cords. The effect of thrush is to cause some discomfort in feeding, local redness, dryness, and possibly pain, slight pyrexia, and considerable impairment of the infant's progress. The treatment should be preventive rather than curative. Scrupulous care in the cleaning and scalding of milk and milk vessels is necessary. After every feed, bottles should be scalded with hot water containing soda bicarbonate, 1 dr. to the pint, and then rinsed at least three times with clean, cold water. In no circumstances should food be allowed to remain in the bottles between feeds. Very gentle sponging of the infant's mouth with glycerin of borax and water in equal parts after feeding should be carried out if thrush is present, but sponging of a healthy infant's mouth is unnecessary and possibly harmful. Gentle purgation helps to improve the child's

general condition and thereby accelerates a cure. Caution is necessary lest confusion should arise between the appearance of thrush and a small deposit of milk-curd. In the latter case the deposit can be gently brushed off, leaving a normal mucous membrane; in the former, more friction is necessary and there is local redness of the mucous membrane.

2. **Acute simple stomatitis and glossitis** results from local injury and from septic infection. When due to the former its severity varies with the extent and virulence of the injury. Acute inflammation following scalding, or the action of corrosive acids, leads to desquamation of epithelium over large areas of the mouth, tongue, palate and fauces, and sometimes œdema laryngis, and may cause suffocation. Treatment consists in measures to reduce the congestion, and includes the use of ice internally and two or four leeches in the submaxillary region.

3. **Ulcers** of the mouth are simple, syphilitic, tuberculous, or malignant.

(a) **Simple ulceration** includes the traumatic and infective forms. Dental ulcers are traumatic, and are seen upon the cheek or tongue as the result of injury against the sharp edge of a broken or septic tooth. Diagnosis depends upon the recognition of the exciting cause, and treatment consists in its removal.

(b) **Aphthous ulceration** is very common. It commences as single or multiple pin-head vesicles upon the tongue, cheek, or palate; these rapidly break down, leaving tiny, shallow ulcers which may be intensely painful. Treatment consists in promotion of general health by exercise, mild purgation, and the administration of iron, arsenic, and hypophosphites, in mild antiseptic mouth-washes, dental hygiene, and, if the ulcers are painful, the careful use of caustic upon their base.

(c) "**Bednar's aphthæ**," so-called, are symmetrical ulcers of the mucous membrane over the pterygoid processes in weakly and neglected infants. In such infants an indolent ulcer is sometimes found upon the hard palate as the result of friction against the teat or baby's thumb and subsequent mild septic infection.

4. **Streptococcal stomatitis** may occur in fibrinous form already mentioned, or as an extension of erysipelas from the cheek, producing considerable painful swelling and severe general disturbance.

5. **Gonorrhœal stomatitis** is seen occasionally. Its diagnosis can only be made by bacteriological investigation, but a suggestion

## STOMATITIS AND GLOSSITIS

of its nature may arise from the existence of gonorrhoeal manifestations elsewhere. *Treatment* with mouth-washes of silver salts has been successful.

**6. Mercurial stomatitis**, from the therapeutic use of the drug, is seen less commonly than in the past, and legislation has practically abolished it among the trade-workers. It is said that it rarely follows the taking of mercury in pill form, but is specially apt to follow its administration by fumigation or inunction. In view of the occasional occurrence of an idiosyncrasy to mercury, great care should be exercised in prescribing the drug to a patient for the first time. The earliest symptom of intolerance is a metallic taste in the mouth, very quickly followed by tenderness of the teeth and gums, passing on to intense congestion and swelling with loosening of the teeth and sometimes hæmorrhage from the gum; at the same time the breath acquires a peculiarly offensive odour, and salivation is profuse. *Treatment* is first preventive, by scrupulous attention to the condition of the teeth before the drug is given, the employment of very small doses till the patient's tolerance is recognized, and insistence upon attention to the teeth and gums during the course of administration. In addition to the affection of the gum which is the most common manifestation of mercurial stomatitis, an acute parenchymatous glossitis is occasionally seen.

**7. Gangrenous stomatitis** (Noma or Cancrum Oris) is a very serious condition; it affects children whose vitality is low, especially during early convalescence from measles or scarlet fever. The process starts in the deeper layers of the mucous membrane of the cheek and spreads with an intense rapidity, destroying all tissue which it reaches; thus extensive sloughing of the cheek, gum, nostril, and orbit may occur. The black, greenish, or greyish gangrene imparts to the breath a characteristic penetrating carrion-like odour. Usually the general disturbance is profound, but cases occur in which it is slight. Death usually occurs from septic broncho-pneumonia. No certainty exists as to the nature of the causal organism; various forms of streptotriches and spirilla have been described, and probably the infection is a mixed one. *Treatment* must be very vigorous. Destruction or removal of the whole of the diseased area by free surgical excision or extensive cauterization is called for. At the same time, the strength of the child must be supported by every available means.

**8. Syphilis** of the mouth appears at any stage. **Primary chancre** in the mouth is not so common as upon the tonsil, but it may occur upon the anterior pillar of the fauces and as a rarity upon the tongue. The chancre quickly ulcerates and is surrounded by considerable inflammatory swelling. The spirochætes can be detected in scrapings from its surface. Secondary symptoms make their appearance after the usual interval.

**Secondary syphilis** affects the mouth in three ways—(a) rash, (b) condylomata, (c) ulceration. (a) The rash is a patchy, brownish erythema upon the hard and soft palate and cheek. (b) *Condylomata* or mucous patches are raised, circular, silvery plaques, sodden in appearance, occurring upon the mucous membrane, especially near the lips; they are intensely infective. (c) *Ulceration* either occurs as the result of breaking down of condylomata or arises spontaneously. Its situation in the latter case is usually in the posterior half of the mouth; the ulcers are shallow, spread and heal slowly, producing a serpiginous outline and leaving a thin white silvery scar which has been aptly compared to snail tracks.

**Tertiary syphilis** affects especially the tongue and palate, though gummatous ulceration of the cheek is seen occasionally. The tongue lesion may be superficial or deep. Chronic superficial glossitis with loss of papillæ and local atrophy or thickening of the epithelium is the most common form. Chronic sclerosis of the tongue as a whole may occur, producing shrinking and stiffening of the organ. Gummata usually arise in the depth of the tongue, and are very rarely multiple; they give rise to little pain until ulceration and septic infection supervene. Gummatous ulceration is a very common manifestation of buccal syphilis; it affects principally the hard and soft palate, leading to destruction and deformity which may be painless.

The *treatment* of buccal syphilis differs in no important particular from that of syphilis elsewhere.

**9. Affections of the gums.**—(a) True **hypertrichy** is an occasional phenomenon; it may be local or universal, and is in some cases of congenital origin.

(b) **Spongy gums** are seen in dental sepsis, mercurial stomatitis, scurvy, and some forms of purpura. Their *treatment* is that of the causal condition, and the local use of astringent antiseptics such as potassium chlorate or weak dilution of hydrogen peroxide.

## STOMATITIS AND GLOSSITIS

10. **Disorders of the tongue.**—(a) **Fur** is due to an accumulation of food *débris*, moulds, and other organisms; it is predisposed to by any condition leading to dryness of the mouth and lessening of friction, as shown by its unilateral distribution in hemiparalysis of the tongue.

(b) **Black tongue** is seen occasionally as a temporary phenomenon in the subjects of wasting diseases. Two factors combine in its production, an hypertrophy of the filiform papillæ at the back of the tongue, and a growth of a black mould. The condition has to be distinguished from blood-staining of the tongue surface after hæmatemesis or concealed nasal or buccal hæmorrhage.

(c) **Geographical tongue**—erythema migrans—is met with not uncommonly in children. On the dorsum of the tongue circular patches develop in which there are enlargement of the fungiform and prominence of the filiform papillæ. Gradually the centre of the patch reverts to its normal appearance, but meanwhile the process has extended at the periphery and probably coalesced with the margin of an adjacent patch. In this way the surface of the tongue acquires an appearance which has a superficial resemblance to spreading ringworm, and accounts for the incorrect name “ringworm of the tongue,” still sometimes employed. The condition is harmless, but very resistant to *treatment*. Its disappearance may follow the use of general tonics and change of air. There is little evidence that it is specially connected with digestive disturbances.

(d) **Lichen** of the tongue occurs as a rarity, usually in a patient who is or has been the subject of cutaneous lichen. The patch varies in size, is usually bilateral, has a glazed, white appearance, gives rise to little discomfort, except a sense of heat, and is resistant to treatment. The condition must be distinguished from leucoplakia, in which the surface of the tongue is harder and more glistening, and from syphilitic superficial glossitis.

(e) **Ulcers of the tongue** are common in unhealthy states of the mucous membrane of the mouth. They are seen in neglected thrush, as a result of irritation from the edge of a broken septic tooth, and as a manifestation of aphthous stomatitis. The various forms of lingual ulcer are considered in TONGUE, ULCERATION OF, and in other articles on diseases of the tongue; but *Riga's disease*, which is very occasionally seen in this country, may be noticed here. It consists of a whitish plaque

## STRABISMUS

upon the frænum of ill-nourished infants. By occlusion it may lead to retention-cysts of the sublingual ducts. It may ulcerate and usually persists for several weeks. The *treatment* of simple ulceration of the tongue resolves itself into general or local treatment of the causal condition and symptomatic treatment of the ulcer itself. Painful ulcers should be touched with pure carbolic acid or silver nitrate. Care must be exercised to prevent spreading of these reagents to the surrounding healthy mucous membrane. Some forms of ulcer, such as those resulting from certain cases of herpes, are so painful that painting with cocaine solution (2 per-cent.) is necessary before food can be taken; the patient must be warned to refrain from swallowing the cocaine-impregnated saliva.

C. E. SUNDELL.

**STOOLS, EXAMINATION OF** (see FÆCES, EXAMINATION OF).

**STOOLS, TARRY** (see MELÆNA).

**STRABISMUS.**—No subject is more important for the general practitioner to understand thoroughly than this, for he is the first to be consulted when parents believe that their children squint, and much depends upon the advice which he gives at this time.

In no circumstances should a squint be left untreated, however slight or transient it may appear to be; no child is too young to be treated on the lines laid down in this article. The earlier the treatment is carried out, the more satisfactory will be the result, and the greater the chances of complete cure with full standard of vision in both eyes. The old notion of waiting till “teething is over,” in the hope that the squint will then disappear, or that the child “will grow out of it,” is fatal to success. Apart from paralysis there is no such thing as squinting with both eyes, and excessive convergence, e.g. so-called “looking at the tip of one's nose,” is not squinting, as is so often stated even by those who ought to know better. Such are the erroneous views which lead to delay in seeking expert advice. It must clearly be understood that a squint exists only when one eye is looking straight ahead while the other assumes or tends to assume an abnormal position, either inwards, outwards, upwards, or downwards; the commonest in children being the inwards position.

**Apparent and real squint.**—The first thing to determine is whether a real squint is present or not.

## STRABISMUS

When both eyes are really fixing an object, and yet one of them appears to diverge or converge, the squint is *apparent*. Two of the principal causes are an epicanthus or a broad bridge to the nose, making the eyes appear nearer to the inner canthus than normal, and a difference in size of the angle which the visual axis makes with the optical axis at the centre of rotation of the eyeball. It may be large, as is often the case in hypermetropia, or small or even negative, as in high myopia; the former gives rise to apparent divergence, and the latter to apparent convergence. It will be noticed that exactly the opposite is true of real squint.

Real squint may be either concomitant, paralytic, or latent; the present article is concerned with concomitant and latent strabismus only.

### CONCOMITANT STRABISMUS

This may be convergent, divergent, upwards, or downwards; the two former are the commonest. Convergent strabismus is usually associated with hypermetropia, and divergent with myopia, but occasionally one meets with true divergence in hypermetropia and true convergence in myopia. In concomitant strabismus the relative positions of the two eyes remain the same, in whatever direction the eyes may be turned.

To discover whether or not a real squint has to be dealt with, direct the patient to fix an object about 2 ft. from the face exactly in the centre of the field of vision. Then determine which appears to be the fixing eye and which the squinting eye. Next, cover the fixing eye with one hand and ask the patient still to look at the object; to accomplish this the squinting eye must make a movement in order to come to the position of parallelism, thus proving that this eye was not fixing before. If no movement takes place there is no real squint but only an apparent one. An exception to this rule is met with when there is complete loss of vision in the squinting eye, which has become practically blind from disuse; nevertheless, the test is accurate and reliable.

**Etiology.**—The two chief causes of squint are—

1. Disturbance of the normal co-ordination of the ocular muscles.
2. Weakness or absence of the fusion-sense; or tendency to use the eyes separately rather than together.

If both these causes are present, a squint is bound to result. When one of them is present while the other is not, certain predisposing causes may produce a squint. For instance, if the muscle-balance is defective and the fusion-sense is good, the eyes are held in position by the fusion-sense; but if the fusion-sense subsequently be lost by one eye becoming defective or blind, the eye at once deviates inwards, outwards, upwards, or downwards. Such cases are examples of latent squint.

If the muscle-balance is good and the fusion-sense defective, the eyes remain straight so long as the muscle-balance is not disturbed; when it becomes deranged by an acute illness, teething, fall, fright, shock, excessive convergence and accommodation as in hypermetropia, or insufficient convergence as in myopia, a squint immediately develops. In most cases the refractive error is the chief predisposing cause, though the others play an important part; in children, for example, the squint often becomes noticeable at the time of teething or after a fall on the head.

Since glasses are ordered for most patients with squint, it is well to remember that the degree of squint bears no direct relationship to the amount of refractive error, some patients with a very low degree of error having marked strabismus, whilst others with a high degree of error show no sign of strabismus at all; the defective muscle-balance and fusion-sense are therefore the main causes of the deformity.

In order clearly to understand this combination of causes, let us take the case of *convergent* concomitant strabismus, which is commonly found in hypermetropia. People with hypermetropia are forced to accommodate to see any object distinctly, even in the distance, and, since accommodation and convergence act in unison, it follows that hypermetropes must also converge for the distance. This constitutes the muscle-balance inco-ordination. Now, the normal position of the eyes for seeing distant objects is one of parallelism, the image being focused simultaneously on both maculae; but if there is convergence the image of the object is thrown on non-corresponding parts of the two retinae. Theoretically, in all such cases diplopia should occur, but in persons whose fusion-sense is well developed the images, which are thus focused only a short distance away from the macula, are fused into one, and no diplopia results. In those with weak fusion-sense, diplopia immediately occurs under these conditions, and can

## STRABISMUS

only be obviated, when both eyes are open, by bringing one eye parallel and throwing the whole of the convergence, usually shared between the two eyes, upon the other eye; this causes the image to be focused in the squinting eye so far away from the macula as to be relatively imperceptible compared with that at the macula in the fixing eye. Convergent concomitant squint is thus produced. If the strabismus remains confined to one eye only for any length of time, the macula in this eye is never utilized, as no image is ever received there; after many years such an eye loses the power of accurate central vision, its sight soon deteriorates, and ultimately becomes lost, never to be regained. This is known as amblyopia; it is clearly important that a squint should be treated early, before amblyopia is added to the disease.

The position of a squinting eye in squints other than convergent can be similarly explained. In young people the commonest form of muscle-balance defect is excess of convergence, but as age advances the power of convergence tends to decrease concurrently with the loss of accommodation. In this way some convergent squinting eyes tend to become straight, but by that time the eye is blind and therefore no double vision is noticed, for, although the eyes are straight, only monocular vision remains. This is the origin of the advice, which only contains a half-truth, that the squint will become straight in time if left alone.

**Treatment.**—In every case of squint the ideal to be aimed at should be perfect vision in both eyes, parallelism of the visual axes, and good binocular vision of the third grade, viz. the estimation of the three dimensions of space; only when this is attained should glasses be left off.

Parents are naturally most anxious about the disfigurement and the inconvenience associated with the wearing of glasses; the defect in the sight is not appreciated by them because, when both eyes are open, the visual acuity is judged by the power of the fixing eye alone; a false impression of good sight in both is thus created.

The treatment of concomitant strabismus must be carried out in a definite order; first the refractive error must be corrected, then, if necessary, the muscle-balance regulated, and lastly the fusion-sense educated. Any deviation from this order may lead to trouble which it may be difficult to rectify. Because the

parents lay so much stress on the disfigurement, they may try to force the practitioner's hand as regards operative interference; it is probable that this temptation to operate prematurely for squint, which was the practice many years ago, is responsible for the cases of divergence which we occasionally see. They could no doubt have been cured by the use of glasses. After the vision in *each eye separately* has been accurately recorded, the amount of refractive error should be measured by retinoscopy under atropine (1-per-cent. atropine ointment or drops three times a day for a week). The practitioner may then, if he has had a fair amount of experience in refraction work, order glasses and carry out the first step in the treatment on his own account. No child is too young to be put into glasses when once a squint has been definitely diagnosed, and the early cases are the very ones which are likely to be cured by glasses alone, without operative interference.

The children must wear the glasses constantly from the moment they get up in the morning till they go to bed at night. If there is any tendency to look over the top of the glasses when, as in hypermetropia, the full correction has been ordered, order large circular frames reaching up to the eyebrow.

The deviation is not always rectified at once, and during the waiting period it is very important to educate the squinting eye by some of the methods now in use. One of the best is to have another pair of glasses made with a well-fitting black goggle opposite the fixing eye, in order to bring the squinting eye into use. If the children are too young to read letters, they may be shown pictures while this pair of glasses is worn; or if there is little power of fixation, they may run about for half the day with the black goggle glasses on. Older children can be made to read letters and books for definite periods each day with the squinting eye alone, the other being blocked up, until it is immaterial to them which pair of glasses they wear; it may then fairly be assumed that the visual acuity of the squinting eye is becoming equal to that of the fixing eye. Another method is to keep the fixing eye under atropine so that it can only be used for distance, while the squinting eye can be used for both distant and near vision; but on the whole I do not think such good results are obtained when both eyes are open as when the fixing eye is entirely covered.

Whatever subsequent treatment may be



## STRABISMUS

carried out, success largely depends on securing equality of visual acuity in the two eyes, and therefore this educative period is very important. It is better to wait at least a year, and sometimes longer, before pronouncing definitely whether the glasses alone are sufficient to bring the eyes straight. If at the end of this time there is no improvement, it generally means that the correction of the refractive error has not been sufficient to overcome the muscular defect, or that the muscles have become fixed or more or less contracted. It is therefore necessary to deal with these muscles by one of the various operations which have been devised, but which should only be considered at this stage.

Operations on the muscles are directed towards weakening the relatively stronger muscle (i.e. tenotomy), or advancing the attachment of the weaker muscle (i.e. advancement), or both. There is much difference of opinion as to which method is the better, for it is difficult to gauge the remote effect of any operation on the ocular muscles, and the permanent result varies somewhat in different cases. Strange as it may sound, the best results are not always attained by getting the eye absolutely straight (as tested by a Maddox rod) at the moment when the operation is completed. All such operations require a considerable amount of experience both as regards technique and judgment in deciding what type of operation is most suitable in a given case; he is a wise practitioner who leaves this part of the treatment of squint to an expert.

As soon as the muscle-balance has been adjusted to the normal, the third stage in the treatment must be begun. This consists in the education of the fusion-sense by means of exercises with the stereoscope or amblyoscope undertaken regularly every day.

No satisfactory education of the fusion-sense can be carried out while there is still any deviation of the squinting eye, except by special mechanical contrivances connected with illumination, with arranging the image on the macula of the squinting eye, etc.

There are three grades of binocular vision to be developed, and various pictures have been devised to enable the patient to acquire these different grades; it is of little use to pass on to the second before the first is mastered, or to the third before the second is well developed.

In the first-grade pictures one half is presented to one eye and the other half to the

other. Well-known pictures are those of a mouse and a cage, or a parrot and a pole; the child acquires the power of putting the two halves into one picture and seeing it as a whole, so that the mouse is in the cage or the parrot on the pole. At first he sees only the mouse or the cage, but not both together.

In the second grade the two separate pictures are presented to each eye, but the inner halves are similar while the outer halves are dissimilar; by practice the child recognizes the whole as one picture, the inner halves being fused into one image.

In the third grade a number of geometrical figures are placed in the stereoscope, and, if seen properly, are recognized as objects or parts of objects having definite form, depth, and shape (such as spherical wire globes, etc.); these after a time the child recognizes as such and not merely as figures on a flat surface, the sense of perspective being thus acquired. When this third grade of binocular vision is reached the squint is cured, the eyes are straight, there is perfect vision in each eye, and the highest ideal has been attained. If a child has third-grade binocular vision, however much the eyes appear to squint, deviation must be apparent and not real.

A convergent strabismus may be *alternating*, where the child squints sometimes with one eye and sometimes with the other. In these cases the vision is generally perfect in each eye from the beginning, but on the other hand the development of the fusion-sense is particularly difficult and often impossible.

*Divergent* concomitant strabismus is found in some cases of myopia where the convergence is specially weak, and a similar line of reasoning as for convergent strabismus may be applied to this form, substituting excessive weakness of convergence for overaction of accommodation and convergence.

It is practically impossible to cure divergence by means of glasses alone; operative measures are always necessary. These consist in advancing the internal rectus and tenotomizing the external; it is generally advisable to aim at leaving a slight amount of convergence after the operation is completed.

**Prognosis** is, on the whole, favourable if the squint is discovered early in life and treatment begun at once. The earlier the squint develops and the longer it is left before advice is sought, the worse the prognosis. When the squint begins after the child is over about 5 years of age, and continues for

## STRABISMUS

several years before it is treated, the prognosis is better than when it begins earlier and remains untreated for the same length of time.

The actual deformity can always be removed by operative measures, though more than one operation may be necessary, but the ideal result mentioned above cannot always be secured in spite of careful treatment carried out under the best conditions.

### LATENT SQUINT

As the name implies, latent squint occurs in cases in which the deviation is not apparent so long as both eyes are open and have equally good vision. Here the muscle co-ordination is defective but the fusion-sense is strong and good, so that any tendency towards the development of malposition due to the former gives rise momentarily to double vision, which is immediately counteracted by an increased stimulation to muscular action prompted by the good fusion-sense, so that the muscle co-ordination is readjusted at once. But this abnormal increase in muscular effort has to be exercised all day, as long as the eyes are open, and in some cases the effort cannot be sustained without causing symptoms. Both eyes must have equally good vision, otherwise the stimulus to binocular vision cannot originate, since the image of objects in one eye is so different from that in the other, and this forms the basis of the various tests which are applied for the detection of latent squint. When one eye is completely blind, there being no stimulus towards muscle co-ordination, this eye occupies a position of deviation in whatever direction the strongest muscle draws it. It is for this reason that some blind eyes pass permanently into a position of convergence, divergence, etc.

The **symptoms** complained of are headaches lasting all day, not specially related to the use of the eyes for near work, and due to the constant strain on the relatively weaker muscles; and temporary diplopia, especially when the patient is tired, owing to a momentary failure of the same set of muscles. In extreme cases, while reading, one part of a line of print may occupy a different level from the other part.

**Diagnosis** is not difficult if the special apparatus for detecting the weakness is at hand. One of the commonest tests is the Maddox rod (Fig. 92), a series of cylinders fixed in a frame so that it can be conveniently placed in front of one eye in a trial frame. If the

rod is of red glass it will produce the image of a red line while observing a small light placed at a distance of 6 metres; if the other eye is kept open at the same time, one eye will be looking at a red line of light and the other at the ordinary white light: the images will therefore be dissimilar, and there will be no stimulus to fusion. If there is no muscular inco-ordination the image of one eye will be superimposed on that of the other, but if there is any latent squint there will be a separation of the images of greater or less degree, according to the power of the muscles which are the stronger, the eyes being drawn into a corresponding position. It is best to place the rod in position so as to bring out the horizontal line in testing the upward and downward movements, and in the opposite position to bring

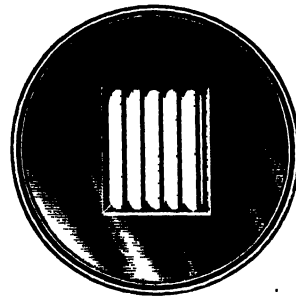


Fig. 92.—Maddox rod.

out a vertical line when testing the external and internal movements. If a Maddox rod is not handy, simply cover one eye with a screen; this will effectually destroy binocular vision; and then it will be noticed that the eye behind the screen has changed its position, to be quickly restored again by a rapid movement of the eye as soon as the screen is removed, in order to counteract the otherwise temporary diplopia which occurs in these circumstances. This movement can be detected if the eye which has been covered is carefully watched immediately the screen is removed.

**Treatment.**—Since latent squint has nothing to do with refractive errors, though these may possibly exist at the same time, the treatment must be carried out on different lines. It consists in the performance of muscular exercises, in the prescribing of prisms, or in operations on the muscles. These cases require a considerable amount of judgment, and should be handed over to an ophthalmic surgeon.

MALCOLM L. HEPBURN.

## STRIDOR

**STRABISMUS, PARALYTIC** (*see* OPTHALMOPLÉGIA).

**STRAMONIUM POISONING** (*see* POISONS AND POISONING).

**STRANGULATION** (*see* ASPHYXIA).

**STRICTURE OF ŒSOPHAGUS** (*see* ŒSOPHAGEAL OBSTRUCTION, under ŒSOPHAGUS, AFFECTIONS OF).

**STRICTURE OF RECTUM** (*see* RECTUM, STRICTURE OF).

**STRICTURE OF URETHRA** (*see* URETHRAL STRICTURE).

**STRIDOR. Etiology.**—The following are the usual causes :—

*In Infants :*

1. Congenital laryngeal stridor.
2. Pressure of an enlarged thymus gland on the trachea.
3. Laryngismus stridulus.
4. Laryngitis.

*In Children :*

1. Multiple papillomata of larynx.
2. Acute oedematous laryngitis, septic or traumatic.
3. Foreign bodies in larynx or trachea.
4. Glottic spasm.
5. Membranous laryngitis.

*In Adults :*

1. Laryngeal stenosis due to growth, simple or malignant.
2. Growths of the pharynx.
3. Laryngitis, septic, syphilitic or tuberculous.
4. Impaction of foreign bodies in larynx or trachea.
5. Bilateral paralysis of the abductors of the vocal cords.
6. Glottic spasm.
7. Narrowing of the trachea, due either to intrinsic or to extrinsic causes.

The **prognosis, pathology and treatment** of stridor depend upon the cause. This is dealt with in other articles. A short description of *congenital laryngeal stridor* is, however, necessary here. It occurs in newborn infants, and is brought about by an extreme narrowing of the supraglottic structures. The epiglottis is folded on itself and the aryteno-epiglottic folds are in close apposition. On examination by direct laryngoscopy the aperture of the larynx will be seen as a narrow slit, and on inspiration it will be noticed that the two sides get sucked together, thus

## STUPOR

giving rise to the stridor. The condition must be diagnosed from laryngitis and laryngismus stridulus. It occurs earlier in life than the latter ; the stridor is more or less continuous night and day, and it does not come on in sudden acute attacks, as does that of laryngismus stridulus. There is no rise of temperature, nor cough, as in laryngitis, and the general condition of the child, as a rule, is good. These cases tend to recover in a year or so unless laryngitis or bronchitis supervenes, when the prognosis becomes grave. The treatment is to maintain the general health.

The **diagnosis** of stridor will depend on the age of the patient, the history of the case and duration of the condition, the nature of the attack, etc. It is useful to remember that stridor due to laryngeal causes is inspiratory, that the head is thrown back and the voice is usually hoarse, whereas in tracheal or extra-tracheal causes the stridor is both inspiratory and expiratory, the head is held forward, and the voice is quite clear. J. GAY FRENCH.

**STRYCHNINE POISONING** (*see* POISONS AND POISONING).

**STUPOR.**—A condition in which there is a marked diminution or absence of the external signs of mental activity, although actual loss of consciousness is not present. It is a syndrome which may arise in several quite distinct forms of mental disorder, and its character and significance are very different according to the nature of the disease in whose course it occurs. It will therefore be subdivided under the following headings, viz.—Simple Stupor, Manic-Depressive Stupor, Catatonic Stupor, Epileptic Stupor, Stupor in General Paralysis.

### SIMPLE STUPOR (ANERGIC STUPOR, ACUTE DEMENTIA)

An exhaustion psychosis, akin to confusional insanity, and the only variety of stupor which can lay claim to be a definite clinical entity, and not merely a transitory episode in the course of some other disorder.

**Etiology.**—This is similar to that of the deliria and other confusional psychoses. There is generally a history of a preceding acute infection, or of an exhausting illness, severe shock or emotional strain. Insanity arising during the puerperium not infrequently takes this form.

**Symptomatology.**—In this affection the stuporose phase is generally preceded by a

## STUPOR

preliminary period of mental confusion, often lasting for some weeks. The confusion gradually deepens until a definite condition of stupor is established. The patient lies inertly in bed, does not speak, and takes no notice of any remarks addressed to him, nor of anything else which is happening around him. He remains in any position in which he is placed, and if a limb is raised and allowed to fall it drops flaccidly upon the bed. There is no resistiveness, and *flexibilitas cerea* is rarely present. The face is expressionless and pallid, the pulse small, the respiration shallow, the extremities cold and sometimes cedematous. Food is not taken spontaneously, but the patient will often swallow without difficulty food placed in his mouth. He passes urine and feces in the bed. There is considerable and progressive loss of body-weight. The stupor persists for a long period, ranging from three or four months to a year or more. It then gradually lessens in intensity, the patient becomes more and more accessible and in touch with his environment, and finally a normal mental state is re-established. During the stage of convalescence transitory phases of confusion are often observed. Subsequent memory for the stuporose period is generally very imperfect.

**Prognosis.**—The outlook is good, and the great majority of cases finally recover, although a duration of at least several months must be anticipated. In rare cases some permanent mental weakness results.

**Treatment.**—The main indications for treatment are the provision of skilled nursing and the maintenance of the patient's nutrition and strength. A day and a night nurse are always required, and it is necessary to emphasize the importance of never leaving stuporose patients alone, as the stupor is liable occasionally to be broken by the carrying out of some sudden impulsive action. This contingency, however, is much more likely to arise in other varieties of stupor than that now under consideration. The general cleanliness of the patient and the condition of the skin require constant care, and massage is of considerable value. Adequate feeding is of the first importance. Milk, eggs, and cream should be given regularly and frequently by spoon or feeding-cup. Forceful feeding is rarely necessary, but must be adopted at once if sufficient nourishment is not otherwise taken. Drugs are not usually required, but paraldehyde or other hypnotics should be given if sleep is defective, although in this condition it is

naturally difficult to ascertain with certainty how much sleep is being obtained. It need hardly be pointed out that the patient must be kept in bed, and that attempts to rouse him to any kind of activity are worse than useless. In long-continued states of stupor, thyroid extract has sometimes been employed, but its value in these cases is a subject of dispute.

**STUPOR IN MANIC-DEPRESSIVE INSANITY.**—A stuporose condition may appear during a depressive stage (*melancholic stupor*), or subsequent to a manic phase (*postmaniacal stupor*). In the former case the stupor is to be regarded as an exaggeration of the difficulty and slowness of movement and mentation characteristic of this condition. It differs from simple stupor in the following points: The patient's expression indicates misery rather than vacuity, he is able to understand what is going on, and even to reply intelligently when addressed, although slowly and with difficulty; there is no subsequent amnesia, and resistiveness is frequently pronounced. Postmaniacal stupor is a condition which sometimes develops at the close of a manic attack. It is generally of short duration, and may be looked upon as a phase of reaction or exhaustion.

**CATATONIC STUPOR.**—In the catatonic type of dementia præcox, stupor is frequently observed, but it has here well-marked characters which serve to differentiate it without much difficulty from the varieties of stupor hitherto described. Resistiveness and negativism are generally pronounced, but are often replaced temporarily by *flexibilitas cerea*, the limbs remaining for indefinite periods in any position in which they may be placed. The patient spontaneously adopts fixed attitudes, and sometimes regularly repeated stereotyped movements occur. The history and the presence of other symptoms of catatonia leave no doubt as to the diagnosis. (*See CATATONIA and DEMENTIA PRÆCOX.*)

**EPILEPTIC STUPOR.** Stuporose phases are occasionally observed in epilepsy, either subsequent to a fit or as a replacement phenomenon. The condition is usually transitory, and rarely persists for more than a few hours. The history suffices to make the diagnosis clear.

**STUPOR IN GENERAL PARALYSIS.**—Stupor in general paralysis is commonly only a transitory phenomenon, but occasionally more prolonged attacks occur. In these states considerable clouding of consciousness is present, and there may be a general resemblance to simple stupor, but the history and the physical

## SUBINVOLUTION OF THE UTERUS

signs serve to differentiate the condition without difficulty.

**Diagnosis of stupor.**—The recognition of the fact that stupor is present is a simple matter. Dementia and profound mental deficiency (idiocy) may superficially resemble it, but the history makes the true nature of the condition obvious. The partial coma met with in cerebral tumour and other gross brain diseases is strictly speaking a form of stupor, but it is not customarily included therewith. In any case, the distinction gives rise to no practical difficulties. The determination of the precise form of stupor present may not always be easy, but the points upon which the differential diagnosis must be based are indicated in the descriptions given above.

**Prognosis and treatment.**—Of the varieties of stupor other than simple stupor the prognosis and treatment depend upon those of the primary disease. In so far as the stuporose condition in itself requires special treatment, this must be carried out along the lines laid down in the case of simple stupor.

BERNARD HART.

**STUTTERING** (*see* SPEECH, DISTURBANCES OF).

**STYE** (*see* EYELIDS, AFFECTIONS OF).

**SUBINVOLUTION OF THE UTERUS.**—

The condition which results when processes of involution, or physiological shrinkage of the uterus after labour or abortion, have not progressed to their full extent in the normal time. After full-term labour complete involution requires about six weeks; by then the uterus should have returned to the state of the normal unimpregnated organ.

**Etiology.**—Involution is brought about by a self-digestion (autolysis) of the muscle-fibres, due to the liberation of a peptonizing ferment, whereby the fibres are reduced in size, though not in number, without any fatty degeneration taking place. For peptonization to occur in due time there must be a normal vascular accommodation, and it is found that any derangement of the blood supply of the uterus or pelvis will retard the process.

While there are many apparent causes which underlie subinvolution, it is probable that the fundamental one is vascular congestion. Hence any condition which predisposes to congestion of the uterus is liable to hinder normal involution. The commonest are—(a) retention of

the products of conception or of bloodclot within the uterus; (b) infection of the uterine cavity or muscle, either by putrefaction of retained products, or by direct streptococcal infection of the uterine tissues; (c) pelvic cellulitis or peritonitis; (d) the presence of a fibro-myoma; (e) getting up too soon after abortion or confinement (probably a potent factor, especially where ordinary hard physical work is resumed immediately); (f) retroversion of the uterus following labour or abortion; (g) persistent constipation and dyschezia; (h) co-existing general disease, e.g. tuberculosis, acute specific fevers, morbus cordis, albuminuria; (i) inability to suckle, which, though not a determining factor, is probably a predisposing cause. Summarizing the various causal conditions, we find that the chief causes of subinvolution *during the lying-in period* are retention of membrane, placenta, or clot; and infection, either sapræmic, septicæmic, or extra-uterine; whereas the commonest causes of *chronic* subinvolution are the presence of a "placental polypus," infection of the endometrium and muscle (endometritis and "chronic metritis"), and too early resumption of hard work combined with obstinate constipation and the habitual use of purgatives.

**Pathology.**—The uterus is enlarged, from the arrest of the process of autolysis of the muscle-fibres. In the later stages there are added a hyperplasia of the muscle and a small degree of fibrosis. Such a condition is difficult to separate from so-called chronic metritis and fibrosis uteri. The endometrium is always thickened, from vascular engorgement and hyperplasia. Another prominent feature is a congestion of the whole uterus. Retroversion, prolapse, a fibro-myoma, or extra-uterine pelvic inflammation is frequently found also. In recent cases a mass of placenta, lightly attached to the wall of the uterine cavity, may be encountered; but in chronic cases this placental fragment often becomes converted into a "placental polypus" by the deposition on it of fibrin, and by a partial organization of bloodclot.

**Symptomatology.**—*During the first fortnight after delivery* the symptoms are those of the causal condition, together with hæmorrhagic lochia, or even secondary post-partum hæmorrhage. Prolonged "after-pains" may be complained of and, rarely, spontaneous pelvic pain. The signs obtained by examination are (a) a uterus abnormally large for

## SUBINVOLUTION OF THE UTERUS

the puerperal date, (b) uterine tenderness on abdominal palpation, (c) abnormal softness of the cervix and an unduly patulous os; (d) the uterus may be displaced backwards and (e) there may be a small fibro-myoma.

In *chronic* cases, some weeks after labour or abortion, the symptoms are rather different. The patient complains chiefly of pelvic pain of a bearing-down nature, and backache, much worse after standing and walking, of uterine hæmorrhage, either metrorrhagic or menorrhagic in type, and of a leucorrhœal discharge between the periods. There also may be some prolapse of the vagina or uterus, irritability of the bladder and constipation. More general symptoms are dyspepsia, headaches, lack of energy, and inability to do ordinary work.

The history is important. The symptoms all date from the labour or abortion. There is usually a description of an abnormal confinement involving a prolonged stay in bed, but, on the other hand, the patient may have resumed work very soon after labour.

**Treatment.**—Cases met with *during the lying-in period* usually need treatment for the cause, such as sapræmia or septic endometritis, by hot vaginal douches, ergot, saline purges, and the Fowler position. When a large piece of placenta has been left within the uterus, it must be removed by the finger, never by the curette.

For *chronic* cases the procedure depends upon whether there are retained products within the uterus. Should this be the case, the cervix must be dilated under anæsthesia and the uterus curetted. This is usually followed by free hæmorrhage, which may necessitate plugging of the uterus. After the operation the patient should be kept in bed for at least a fortnight, or longer if there be bleeding, ergot being given throughout the convalescence.

If, from the slight degree of enlargement and the presence of a closed cervix, there be no reason to suspect retained products, treatment may be restricted to confinement to bed for two or three weeks, with hot vaginal irrigation and full doses of ergot combined with quinine. If, after the resumption of ordinary work and routine, hæmorrhage and backache return, curettage should be employed.

A. W. BOURNE.

**SUBMUCOUS FIBROIDS** (see UTERUS, NEW GROWTHS OF).

## SUBPHRENIC ABSCESS

**SUBPHRENIC ABSCESS.**—A localized collection of pus in contact with the under-surface of the diaphragm. It may lie within the peritoneal cavity or in the retroperitoneal tissues. The *intraperitoneal* form is the more frequent, and four varieties are recognized, according as to whether the pus lies to the right or left of the falciform ligament and in front of or behind the corresponding lateral ligament of the liver. Thus, a *right anterior intraperitoneal subphrenic abscess* will lie between the upper surface of the right lobe of the liver and the diaphragm in front of the right lateral ligament; a *right posterior abscess* lies behind the right lateral ligament in what is commonly known as the subhepatic or right kidney pouch; a *left anterior subphrenic abscess* lies between the diaphragm above and the left lobe of the liver and the stomach below; while a *left posterior abscess* is one which occupies the lesser sac of the peritoneum. **Retro- or extraperitoneal** subphrenic abscesses are spoken of as right or left, according as they encroach upon the space between the right or left lateral ligaments.

**Etiology and pathology.**—The causes of subphrenic abscess are varied. The commonest precursor is disease of a viscus lying in contact with the under-surface of the diaphragm—stomach, duodenum, liver, gall-bladder, or kidneys. Of distant organs, disease of the appendix vermiformis is the most important in this connexion, but there appears to be no abdominal viscus which may not be concerned. Diffuse peritonitis in such exceptional cases as do not prove rapidly fatal is sometimes responsible, residual collections of pus accumulating in the neighbourhood of the liver and spleen. A localized tuberculous peritonitis is another infrequent cause. Disease of the upper lumbar vertebræ is accountable for some cases. It is noteworthy that whereas an empyema not infrequently occurs as a secondary complication to a subphrenic collection of pus, it is unusual for a subphrenic abscess to arise secondarily to an empyema. The explanation is to be sought in the direction of the lymph-flow, and possibly in the fact that whilst the intrathoracic pressure is negative the intra-abdominal is positive.

An intraperitoneal abscess lying to the *left* of the falciform ligament starts most commonly from a perforating ulcer of the stomach. When an ulcer of the stomach slowly erodes the wall of that organ, adhesions to neighbouring structures not infrequently form before

## SUBPHRENIC ABSCESS

the final perforation of the peritoneal coat takes place. This condition is sometimes called "chronic or latent perforation," and the process results in the formation of a perigastric abscess. At first the limits of the abscess are determined by the surrounding adhesions. Such a subphrenic abscess will contain not only stomach contents mixed with pus, but also gas. In more than half the cases of subphrenic abscess gas is present. The gas may be derived, as in the above instance, from the giving way of the wall of a hollow viscus—hence gas is more common in left-sided subphrenic abscesses or less frequently from the activity of gas-forming bacteria such as *B. coli*. It is for this latter reason that a right-sided subphrenic abscess consequent upon a suppurative appendicitis not very infrequently (one out of every six or seven cases) contains gas. Suppuration in the lesser sac of the peritoneum (left posterior intraperitoneal subphrenic abscess) most commonly results from a perforating ulcer on the posterior wall of the stomach. It is the rarest variety of all.

An abscess lying to the *right* of the falciform ligament usually originates in connexion with the appendix vermiformis, liver, gall-bladder, duodenum, or right kidney, and only exceptionally in connexion with the stomach. Either an intraperitoneal (anterior or posterior variety) or an extraperitoneal abscess may result when inflammation spreads from the appendix, according as to whether the pus tracks upwards within the peritoneal cavity along the outer side of the colon, or extends by way of the loose cellular tissue behind the ascending colon. Intrahepatic suppuration is the most frequent cause of a right-sided extraperitoneal abscess, while in Barnard's series of cases periostitis of the transverse process of the first or second lumbar vertebra was the most common cause of the rare left-sided extraperitoneal abscess.

The character of the pus from a subphrenic abscess varies with its source. The pus may be admixed with stomach or intestinal contents, bile, urine, with anchovy-like material derived from a liver abscess, or with daughter cysts from a hydatid. The bacteria most commonly met with are *B. coli* and staphylococci. Not infrequently the pus is sterile.

**Symptomatology and diagnosis.**—Recognition of the existence of a subphrenic abscess may be a matter of great difficulty. In rather more than half the cases the onset is sudden. The initial symptoms in many cases

are those of perforation—sudden pain, vomiting, etc.—and as localization occurs these subside. In other cases the onset is insidious, and this is true generally of the extraperitoneal abscesses. When a failure of recovery, after surgical intervention in cases of disease of abdominal viscera, is found to be associated with the persistence or reappearance of fever, a maintained high pulse-rate, increasing pallor, and eventually wasting, the possibility of subphrenic suppuration should be kept well in view. As with abscesses in other situations, there may be hyperæsthesia of the skin and tenderness on light or deep pressure. In some cases vomiting is a prominent feature. The patient soon passes into the toxæmic state, the tongue becomes furred and dirty, the complexion muddy, rigors and sweats may be present, and the chart shows a remittent type of temperature. Blood-examination reveals the existence of leucocytosis. As the case proceeds, increasing listlessness, shortness of breath, and diarrhœa are often noticed. The patient not infrequently complains of pain in the neighbourhood of one or other shoulder, and, as Cope has recently pointed out, valuable indications as to the localization of the pus may be obtained by attention to the position of the referred pain. Thus, pain is commonly complained of in the isolateral subclavicular region when pus is situated towards the front of the diaphragm, pain over the back of the shoulder and supraspinous fossa suggests pus towards the back of the diaphragm, and pain over the acromion has been present with pus in a midway position.

Locally, inspection may show some prominence in the upper abdomen, and possibly a limitation of abdominal movement. In some cases unilateral limitation of the thoracic movements may be detected, and measurement may reveal an increased circumference on the affected side. Palpation not infrequently elicits tenderness over the lower ribs. Any swelling that may be felt in the abdomen is often ill-defined and does not descend on respiration because its lower edge is usually limited by adhesions to the anterior abdominal wall. The liver is rarely displaced downwards unless intrahepatic suppuration has occurred. In left-sided cases the heart's apex-beat may be found displaced upwards. Abscesses containing gas are usually easier to detect than those which do not. If gas be present, percussion shows the existence of three or four zones varying in pitch and resonance.

## SUBPHRENIC ABSCESS

Starting above, percussion will elicit normal lung resonance; below this, should the pleura contain fluid, a zone of dullness will be encountered; lower still, a zone of resonance or even tympany due to the accumulation of gas within the abscess will be found; while at a still lower level, dullness due to pus and merging on the right side into liver-dullness may be detected. A bell-sound over the tympanitic area may perhaps be elicited, and, when the resonant area is high up in the epigastrium or over the area of normal liver-dullness, it may be possible, by rolling the patient over on to his side, to demonstrate that the tympanitic area changes its position *pari passu*, but usually this can only be done when the amount of gas present is large. With no gas in the abscess, and fluid present in the pleura, the existence of a subphrenic abscess becomes more difficult of recognition. The patient is often too ill to undergo radiographic examination, but when this is not the case such an examination may be of great assistance. If a subphrenic abscess exists the diaphragm will be found elevated and immobile, whereas in a case of uncomplicated basal empyema the diaphragm is immobile and depressed; and the heart, too, may show some lateral displacement. An exploring needle may be necessary to establish the diagnosis. So obscure may conditions be that even at operation a subphrenic abscess has been mistaken for an empyema. In collections of pus beneath the diaphragm it is frequently noticed on paracentesis that the maximum flow occurs during inspiration, the descending diaphragm compressing the fluid. In tapping an empyema, on the other hand, the maximum flow takes place during expiration, at which time the thoracic cavity is lessened. Exploration in a supposed case of subphrenic abscess, if employed at all, is preferably carried out under anaesthesia, and all preparations must be made to follow the discovery of pus by immediate operation. If this is not done there is grave risk of infection of the peritoneal and pleural cavities. It is not rare to find clear fluid in the pleural cavity, but this must not be allowed to mislead; on pushing the needle farther inwards the diaphragm is perforated and pus will escape should an abscess be struck.

*Malignant disease of the liver* is the only non-suppurative condition likely to be mistaken for a subphrenic abscess. Wasting is common to the two conditions, and pyrexia is not infrequent in a new growth of the liver.

## SUFFOCATIVE CATARRH, ACUTE

On percussion, in the case of the neoplasm there will be an absence of the dome-shaped upper limit of dullness so commonly found in subphrenic abscess, while jaundice is comparatively rare and slight when it occurs in subphrenic suppuration.

**Treatment** is purely surgical. At first the inflammatory exudate is diffuse, and soon becomes turbid with leucocytes. Some surgeons are of opinion that the best time for operation is after the eighth day, by which time a barrier of adhesions is likely to have formed, but most would agree that the abscess should be opened and drained as soon as ever the diagnosis has been made. In exploring, a deep puncture is made in the 10th, 9th, 8th, and 7th spaces, in this order, first in the scapular and then in the mid-axillary line. Once the needle has perforated the diaphragm, it will usually be found to move with the respiratory movements. When pus is found, the rib below is usually resected, the edges of the diaphragm are sewn to the intercostal muscles, and the abscess is drained. The mortality of cases operated on is about 30 per cent. It is usually stated that all the patients die who are not treated operatively. The abscess may rupture into the pleural cavity, lung, peritoneal cavity, or intestine in cases in which surgical aid is not employed.

C. E. LAKIN.

**SUBUNGUAL EXOSTOSES** (*see* EXOSTOSES).

**SUDAMINA** (*see* SWEAT-GLANDS, AFFECTIONS OF).

**SUFFOCATION** (*see* ASPHYXIA).

## SUFFOCATIVE CATARRH, ACUTE.—

An acute affection of the large and small bronchi producing severe dyspnoea and cyanosis of rapid development.

**Etiology.**—The condition is rarely met with; when recognized it is usually in adults. Nothing definite is known of its causation. There is a close resemblance between this affection and that resulting from inhalation of irritant vapour, such as bromine and iodine. In cases which have been investigated various organisms have been detected, of which the pneumococcus, frequently associated with other microbes, is generally present.

**Symptoms.**—There is a rapid onset, with extreme dyspnoea and cyanosis. Cough is not a marked symptom. Expectoration, if present, is thin and watery and not abundant. The



## SUNSTROKE

temperature is moderately raised and the pulse-rate correspondingly increased. Breathing is laboured, but resembles that neither of asthma nor of pneumonia. In the course of a few days expectoration is increased, becoming mucopurulent but not abundant. The cyanosis passes off gradually. Signs suggesting scattered broncho-pneumonia may be present.

**Prognosis** is uncertain and largely depends on the general strength of the patient.

**Treatment.**—As so little is known of the pathology, treatment is largely symptomatic. The patient should, of course, be put to bed and allowed to rest in the position which is most comfortable for him. Purgatives should be given. Oxygen should be administered; bubbling it through warmed alcohol is the most efficient method. It should be given for a period of ten or fifteen minutes with a corresponding interval, the facepiece being held as near the patient's mouth and nose as can be tolerated. Belladonna or stramonium may be tried, and adrenalin chloride (10 min.) in extreme cases would be justified. The patient's strength must be maintained by careful feeding at regular intervals, and encouragement and assistance given to him in his struggle to breathe. When the most serious symptoms have abated, treatment as recommended for acute bronchitis should be employed.

CHARLTON BRISCOE.

**SUGGESTION** (*see* PSYCHOTHERAPY).

**SULPH-HÆMOGLOBINÆMIA** (*see* CYANOSIS).

**SULPHURETTED HYDROGEN POISONING** (*see* POISONS AND POISONING).

**SULPHURIC ACID POISONING** (*see* POISONS AND POISONING).

**SUMMER DIARRHŒA** (*see* DIARRHŒAL DISORDERS OF INFANTS).

**SUNSTROKE** (*syn.* Heatstroke, Insolation, Heat-apoplexy, Thermic Fever, *Coup de Soleil*, Siriasis (after Sirius, the dog-star), Sun-traumatism).—An acute condition developing in the presence of the sun's rays and a high atmospheric temperature, accompanied by mental symptoms, hyperpyrexia and, it may be, pulmonary congestion.

**Geographical distribution.**—Sunstroke occurs in the tropics and also during unusually "hot spells" in temperate climates. It is common, for instance, in the big cities of North America during the intense summer heat.

During the great war this condition came more into prominence in Mesopotamia than in any other theatre. Dangerous cases were not commonly met with in Egypt, Palestine, or East Africa, but a few were reported from Macedonia.

**Etiology.**—There has been some confusion with the condition known as *heat-exhaustion* or *heat-prostration*, which must be clearly distinguished from sunstroke. Heat-exhaustion is really dependent upon "heat" more than upon the actinic rays of the sun, and is a syncope attending physical exhaustion in a hot climate; thus it affects heavily-laden soldiers on the march, and is common in stokers, especially in the Red Sea. Sunstroke is probably dependent upon the action of the ultraviolet actinic rays on the cranium.

Sunstroke is apparently caused by an auto-intoxication through retention of certain poisonous products in the body and possibly by the failure of the mechanism by which heat escapes from it. A number of theories have been advanced to account for it, but the experimental work so far has been inconclusive.

**Pathology.**—Rigor mortis appears early and is one of the most striking features at autopsy; the blood is dark and does not clot easily; it is acid in reaction. The number of red cells is said to be increased, and the serum is turbid. The temperature of the cadaver continues to rise after death and may even reach 114° F. Ecchymoses of the skin are often present, and there are petechial hæmorrhages on the heart-valves and on the pleura; free blood may actually be present in the pleural cavities, together with congestion and cedema of the lungs. The nervous system shows marked changes, such as cedema of the brain and leptomeninges with great congestion, and on microscopic examination necrotic changes in the ganglion cells, with chromatolysis of the nuclei and disappearance of Nissl's granules. The cerebro-spinal fluid is clear and increased in amount. In the viscera, hyperplasia and congestion of the spleen and cloudy swelling of the liver and kidneys are present. Cortical changes in the suprarenal bodies have been described.

**Symptomatology.**—Though generally sudden in onset during exposure to the sun's rays, sunstroke is often preceded by prodromic symptoms, such as pains in the limbs, vertigo, headaches, præcordial pains, a hot dry skin, irritability of the bladder, a curious intolerance of light associated with its usual phenomenon

## SUNSTROKE

and an irregular pulse. These are succeeded by hyperpyrexia, often reaching 108 or 109° F., and restlessness, passing on to wild delirium and coma. Three clinical forms have been distinguished by Rho, viz. (1) the *cardiac* or *syncopal* form, with slight pyrexia, pallid face, blanched skin and cold extremities, a thready pulse and irregular breathing; (2) a *cerebro-spinal* or *meningeal* form, with hyperpyrexia, congested face and full rapid pulse; (3) a *pulmonary* or *asphyxial* form, with pyrexia, hot skin, suffusion of the eyes, very rapid pulse and loud stertorous breathing. The condition may last a few hours, or a fatal termination may be delayed for four days. Before death, epileptiform or tetanoid convulsions, excited by the slightest stimuli, may be present. The pupils are contracted until immediately before death; they then become dilated, the mydriasis being associated with incontinence of urine and faeces. The skin of the patient is said to emit a mousy odour. The urine is scanty and may contain albumin and casts, and even red and pus cells.

The milder cases are accompanied by headache, intolerance of light and sound, intermittent vomiting, and restlessness. After recovery from the acute attack many nervous manifestations may remain; persistent headache, irritability alternating with fits of depression, photophobia, intolerance of the sun's rays, various pareses, and tremor are the most common.

After very severe sunstroke, epileptiform convulsions, loss of memory, blindness, deafness, and insanity may supervene.

The headaches are accompanied by marked rises of blood-pressure up to 150 mm. Hg and by a bradycardia of 50–60 beats per minute.

**Diagnosis.**—Genuine sunstroke, even in the tropics, is a much rarer malady than one would suppose, and a common error is to mistake *malaria*, either primary or secondary, for the "effects of heat." This is particularly so in a primary attack of subtertian malaria, in which cerebral manifestations with hyperpyrexia are very common. It is possible, too, that great heat is a potent predisposing cause in the production of these "pernicious attacks." A blood-examination with the object of excluding malaria should invariably be undertaken. It is necessary to be on one's guard concerning *alcoholism*, always bearing in mind that the intemperate are more susceptible than others to the sun's rays. From *cerebro-spinal fever* sunstroke is distinguished by the stiff neck, unequal pupils and Kernig's sign; from *heat-exhaustion*,

by the history of exposure to the sun, by the hot dry skin and hyperpyrexia.

**Treatment.**—The patient must be placed in a cool shady place, the clothing loosened, and, if asphyxia be present, artificial respiration resorted to, and persisted in if necessary for a couple of hours. To reduce the temperature the patient must be placed in a cold bath, or wrapped up in a damp sheet in an airy place. When ice can be procured the skin may be massaged with it, or the patient placed in an iced bath. A practical method is to spray him from the head downwards for a minute or two with ice-cold water from a distance of 20 inches. Ice-cold enemata are also recommended. Care must be taken to stop cold applications when the rectal temperature registers 102° F., or fatal collapse may ensue. After the cold bath, hot-water bottles should be applied to promote perspiration, which is a favourable sign. Should the condition relapse, recourse must be had at once to ice.

In the asphyxial cases venesection should be resorted to, a pint or more of blood being removed with the object of getting rid of the toxic products.

For the cardiac condition caffeine and strophanthus should be given. Chloral enemata are useful in delirious cases.

With a view to relieving intracranial pressure, Rawling has performed subtemporal decompression with success; a simpler method is lumbar puncture, which should be performed in serious cases.

**Prophylaxis.**—In the tropics a suitable sun-helmet with neck shade should be worn; spinal pads are generally advised, although their efficacy is open to question. Green or amber-tinted glasses protect the eyes from the sun's rays, and are of considerable value. Naturally, the clothing should be as light and airy as possible.

P. MANSON-BAHR.

**SUPPRESSION OF URINE** (*see URINE, VARIATIONS IN AMOUNT OF*).

**SUPRA-ORBITAL NEURALGIA** (*see NEURALGIA*).

**SUPRARENAL GLANDS, PHYSIOLOGY AND AFFECTIONS OF.**—The suprarenal glands lie in close relation to the upper poles of the kidneys. Like the pituitary gland, the suprarenal is formed of cells derived from two distinct sources, and in certain animals, as in fishes, the component parts remain distinct throughout life. Thus the medulla, the central

## SUPRARENAL GLANDS, PHYSIOLOGY AND AFFECTIONS OF

and smaller portion of each gland, is composed of cells that are derived from nerve-tissue, whereas the cortex that constitutes 90 per cent. of the gland is formed of tissue derived from the mesoderm, and is of glandular nature.

The cortical portion of the suprarenal is pale, and contains numerous granules of a lipoid nature, whereas the medulla is of a reddish hue and possesses peculiar affinity for chrome stains, an attribute that has given the name chromaffin tissue not only to these cells but also to other structures found in various parts of the body. The chromaffin system in man embraces not only the medullary portions of the suprarenals but also the carotid gland, the coecygeal body, accessory suprarenals when they are present, cells in the sympathetic ganglia, especially in the abdominal and splanchnic ganglia, and certain cells in the anterior lobe of the pituitary.

The suprarenals are ductless glands and produce secretion, probably both in cortex and medulla, which is poured forth into the blood-stream and carried over the body. The secretions from the two parts probably have different functions.

The glands are necessary to life, and loss of both suprarenals is followed, generally at a short interval, by death. Although there is much that is unknown concerning the suprarenals, the following beliefs are generally held: The function of the medulla, together with the chromaffin tissue in other sites, is to produce a definite substance, *adrenalin*, which is continuously being poured forth into the blood-stream. This active principle, which not only can be isolated from the suprarenals in a pure form, but can also be prepared synthetically, is sometimes known as *suprarenin*, *epinephrin*, or *hemisine*, but the original name of *adrenalin* is preferable. It has the chemical formula  $C_9H_{13}NO_3$ . The secretion produced by the cortical cells is more intangible, and no specific constituent has been isolated. The presence of the lipoid granules or cholesterol-ester in such large quantities is only paralleled by their occurrence in the central nervous system, and so it has been suggested that they are in some way essential to the functioning of the central nervous portion of the gland.

The effects produced by the internal secretions of the suprarenals are known with considerable certainty in respect to the active principle *adrenalin*. This has the property of causing catabolic processes throughout the body, the effect upon any tissues or organ being similar to that produced by stimulation of the sym-

pathetic nerves supplying them. *Adrenalin* therefore acts upon all the tissues receiving sympathetic-nerve supply; thus it causes a vaso-constriction with rise of blood-pressure, affecting especially the splanchnic area, contraction of uterine muscle, relaxation of the intestines with contraction of the ileo-colic sphincter, and generally a slowing of the heart-beat as the result of central vagal stimulation caused by the rise in blood-pressure.

Langley has shown that *adrenalin* does not act directly upon the sympathetic nerve-terminations, nor upon the tissue-cells, but upon the so-called receptive substance that lies between them. Just as one of the effects of sympathetic stimulation is to produce an increased sugar supply for the muscles which enables them to contract more efficiently, so it was shown by Blum that intravenous or subcutaneous injection of *adrenalin* produces a condition of hyperglycemia and glycosuria, which continues as long as there is excess of *adrenalin* in the blood. The sugar comes from the stored-up glycogen in the body, and ceases to appear in the urine as soon as that source is exhausted.

These are the main effects produced by the active principle of the medulla, *adrenalin*. Whether the cortex takes part in the formation of *adrenalin*, or produces an active physiological principle of its own, is not definitely known, but it is believed by some that it forms a substance that neutralizes toxins produced during muscular contraction, and by others that it is connected with the development of the genital glands and the determination of sexual characteristics in adolescence.

Pathological changes in the glands producing visible macroscopical lesions, or functional disturbances of the glands caused reflexly by nervous impulses, may modify their activity in two directions. An excessive secretion may ensue, producing the condition known as *hyperadrenia*; or its reverse, *hypoadrenia*, with some degree of failure of hormone production, may result. Each of these conditions will be considered in turn.

### 1. DISEASES ASSOCIATED WITH INCREASED SUPRARENAL SECRETION

An excessive secretion from the suprarenals may occur in association with diseases of other ductless glands. Thus in Graves's disease the amount of *adrenalin* in the blood may be as much as eight times greater than normal. Stimulation of the sympathetic nerves running in the splanchnics to the glands increases the

## SUPRARENAL GLANDS, PHYSIOLOGY AND AFFECTIONS OF

output of secretion, especially from the medulla. The glands may enlarge as the result of a simple hyperplasia; and this in many cases affects chiefly the cortex. In the early stages of tumour-formation the amount of secretion poured forth into the blood may be increased.

The chief tumours that occur in the suprarenals are adenoma, sarcoma, secondary carcinoma, neurocytoma and hypernephroma.

Clinically, the malignant suprarenal tumours, among which are classed the hypernephromata, are the most important cause of clinical states in which there are symptoms of hyperadrenia.

**Hypernephroma.**—This tumour is generally believed to originate in the suprarenal, and often gives rise to secondary metastases in the lungs. It may occur under the capsule of the kidney, and then is believed to be formed from islets of suprarenal tissue lodged there, the so-called "adrenal rests." In a certain number of cases they appear shortly after birth or in childhood, in others they do not occur until adult life has been reached. The **symptoms** are local and general. The local ones are those of a tumour in the region of the kidney. This may be on either side of the body, and does not generally grow to a very large size. It may give rise to a moderate amount of aching in the loin, and in some instances, owing to invasion of the pelvis of the kidney, hæmaturia and renal colic, caused by the presence of clots of blood in the ureter, may occur. Generally, however, hypernephromata do not invade the renal pelvis, and so hæmaturia is not caused. In some instances the abdomen is distended with fluid.

In addition to the local signs and symptoms caused by the tumour-formation there are certain distal changes that are believed to be due to an increase in the secretion of the suprarenal cortex. Thus, in the case of a child a few years old with a hypernephroma, the genital organs in a female may assume the size and shape and maturity of a girl of 18, together with an abundant growth of pubic hair. The skin is often coarse, and pigmentation may be present. Girls may become extremely obese, whereas the change in boys is to a state of burliness and muscularity, with a copious growth of hair upon the face and body.

For the **pathology** of hypernephroma, *see* KIDNEY, TUMOURS OF. The **diagnosis** of a case that shows a definite tumour apparently of renal origin, together with secondary sexual changes, is not difficult, but apart from the latter it may be impossible to arrive at a cor-

rect diagnosis. The **prognosis** is bad, for the disease possesses the characteristics of malignancy, and secondary deposits in the lungs and liver are very prone to occur. Owing to this unfortunate feature, operation is not often successful, so that only palliative measures remain.

In adults also malignant tumours of the suprarenals may occur; they may attack one or both glands, and are characterized by their rapid growth and tendency to secondary dissemination. Apart from the local effects of tumour-formation, hirsuties is very liable to occur. Cases have been recorded in women, whose faces were covered with hair, involving the necessity of daily shaving; in one case, after removal of the tumour, the hairiness disappeared and the face was restored to its normal smooth and delicate appearance.

It is well known that during the climacteric troublesome symptoms occur, as flushings, headache, dizziness, and often increased blood-pressure. It is possible that these are produced by a diminution in activity of the ovaries causing an increased functioning of the suprarenals, with a consequent hyperadrenia, but this inter-relationship is still to a large extent a matter of speculation.

In certain cases of glycosuria it is very probable that the suprarenal is at fault, producing an excessive secretion. Experimentally, it has been shown, as mentioned above, that intravenous injection of adrenalin produces glycosuria, and that the condition may also be induced in some animals by painting the pancreas with the active principle. Clinically, it is possible, as suggested by Langdon Brown, that certain cases of glycosuria occurring transitorily in men suffering from shell shock are due to this cause, and also, possibly, other cases of glycosuria found in older men with a high blood-pressure and arterio-sclerosis.

### 2. DISEASES ASSOCIATED WITH DIMINISHED SUPRARENAL SECRETION

**Addison's Disease.**—This disease is the keystone of suprarenal physiology and pathology. It is characterized by (a) pigmentation, described by Addison as a "singular dingy or dark discoloration of the skin," (b) asthenia, (c) low blood-pressure, (d) nausea and vomiting.

**Etiology, pathology, and symptomatology.**—The cause of the disease is some lesion interfering with the chromaffin system and preventing the formation of its specific secretion in

## SUPRARENAL GLANDS, PHYSIOLOGY AND AFFECTIONS OF

sufficient amount. A lesion is therefore generally found in the suprarenal glands, but in some cases of Addison's disease post-mortem examination has shown these glands to be intact, whereas the splanchnic ganglia have been involved in pathological processes.

The condition of hypoadrenia resulting in the syndrome of symptoms known as Addison's disease may arise suddenly or gradually. In cases of rapid onset it is generally found that there has been an acute hæmorrhage into one or both glands, or else a process of suppuration. The usual onset is insidious, and in these cases the most frequent lesion is a tuberculous infiltration and degeneration of the suprarenal. Other lesions that may occur are new growths, gummata, chronic fibrosis or simple atrophy. In Addison's original description, published in 1855, he points out that it is not necessary for both glands to be affected for symptoms of the disease to be produced. Of the 11 cases on which his original description is based, in 7 both glands were affected, and in 4 only one. Tuberculosis was the lesion in 5 cases, carcinoma in 3, fibrosis in 2, and an acute hæmorrhage in 1. In other words, 45 per cent. of the subjects of the disease, as originally described, suffered from tuberculosis, and 27 per cent. from carcinoma.

The symptoms of Addison's disease are believed to be due to a hypoadrenia. The deficiency of adrenalin in the blood-serum would account for the low blood-pressure. The effect of stimulation of the sympathetic nerves supplying the stomach is to cause an inhibition of its movements, and so lack of inhibition caused by deficiency in adrenalin might account for some of the gastric disturbance. The muscular weakness may be due to a diminution in the glycogen supply to the muscles, owing to a hypoglycæmia, or to the fact that toxins produced by contraction are not neutralized by the cortical secretion of the suprarenals. The pigmentation may be due to an error of metabolism whereby the final stages in which tyrosin is converted into adrenalin are not completed and tyrosin and allied bodies now accumulate in the tissues.

Deposition of melanin occurs, either in patches or universally distributed, especially on the face and hands, in the axillæ, around the nipples, navel, and in the pubic region. It is also found where there is cutaneous pressure, so that a zone may be seen corresponding with the site of the waist-band or of the garter. The mucous membranes of the mouth, conjunctivæ, anus,

and vagina may also show deeply pigmented spots. (See PIGMENTATION.) In some cases there are areas of leucoderma, and Addison noticed that not only the skin but the hairs growing from it are sometimes bleached. There is great vascular asthenia, the heart-beats have diminished force, and palpitation is common. The blood-pressure is low, below 100 mm. systolic pressure, and often lower than 80 mm. The abdominal symptoms consist of some soreness in the epigastrium, together with severe and often persistent vomiting. The vomit is generally watery fluid, but in some cases is tinged with blood. Diarrhœa also is sometimes seen. The gastro-intestinal symptoms, although forming part of the recognized syndrome of the disease, are not by any means constant, and a case may proceed to its fatal issue without them. Although anæmia was one of the features on which Addison laid stress, it is not really a common complication.

The **diagnosis** in typical cases is not difficult, though the vomiting may suggest malignant disease of the stomach, the asthenia combined with pigmentation pernicious anæmia, and the discoloration of the skin any of the other numerous causes of pigmentation.

*Malignant disease of the stomach* may be excluded by lack of any confirmatory evidence, such as a palpable tumour, a dilated stomach, or presence of secondary deposits elsewhere. Examination of the stomach contents chemically and bacteriologically, and an X-ray examination with an opaque meal, are useful adjuncts. It must always be remembered, however, that carcinoma ventriculi and Addison's disease may be coexistent.

*Pernicious anæmia* is readily excluded by a blood-examination.

The common causes of *pigmentation* can be recognized by their characteristics; such are pregnancy, sunburn, drugs, abdominal growths, hæmochromatosis, ochronosis, etc. (see PIGMENTATION). The finding of a low blood-pressure is, generally, valuable confirmatory evidence of the presence of a suprarenal lesion.

The **prognosis** is bad, nearly all the cases proceeding to a fatal termination within a period varying from weeks to several years. In one or two cases a recovery with apparent cure has been described.

**Treatment** is unsatisfactory. Adrenalin may be administered by the mouth in the form of 10-20 min. of the 1-in-1,000 solution twice daily, or 2-20 gr. of the desiccated extract of

## SUPRARENAL GLANDS

the whole gland, three times a day. The effects of adrenalin administration are generally very transitory. Surgical procedures involving transplantation of the gland in man have not yet reached a stage to hold out hope of a cure, although it is possible that great advances may be made along these lines in the future. Apart from these specific measures, treatment consists in keeping the patient in bed and in relieving the symptoms as much as possible. The gastro-intestinal ones are often the greatest trouble, but in some cases the vomiting is rendered tractable by the use of ice internally or by the inhalation of oxygen at frequent intervals, as for ten minutes every hour.

Other conditions in which there is hypoadrenia do not form so distinct and unmistakable a clinical entity as we meet with in Addison's disease.

Thus, in certain cases of congenital hydrocephalus a complete absence of the medulla of the suprarenals has been found.

Infantilism may be associated with a diminution in the amount of the suprarenal cortex, which, as we have seen, normally forms the greater part of the gland.

In some of the acute fevers, such as diphtheria, typhoid, scarlet fever, and cholera, it is believed that the manifestations of cardio-vascular insufficiency are due to a hypoadrenia.

Sudden death in status lymphaticus has also been attributed to a hypoplasia of the suprarenals, and a similar condition has been described in osteomalacia, the pernicious vomiting of pregnancy, and rickets. Finally, it is possible that some types of neurasthenia are caused by a functional disturbance of the suprarenals, whereby their internal secretion is diminished in amount. The various conditions that are from time to time described as due to suprarenal hypoplasia have been mentioned to emphasize what far-reaching results may possibly result from endocrine-gland derangement, but clear, striking, and emphatic proof is not forthcoming even to the extent that it is in Addison's disease, and the matter is largely one of speculation.

G. E. BRAUMONT.

**SURGICAL HÆMORRHAGE** (see HÆMORRHAGE, SURGICAL).

**SURGICAL KIDNEY** (see PYELONEPHRITIS AND PYELITIS).

**SURGICAL SHOCK** (see SHOCK AND COLLAPSE).

## SWEAT-GLANDS, AFFECTIONS OF

### SWALLOWING, DISTURBANCES OF.

—Swallowing may be affected by any disease that narrows the pharynx or œsophagus, as stenosis or a tumour. Occasionally pharyngeal spasm, either reflex or functional, interferes with the passage of food, but the most common cause is palsy of the pharyngeal muscles. These are innervated through the pharyngeal plexus by branches of the vagus and glosso-pharyngeal nerves.

Unilateral palsy rarely produces serious symptoms, but complete bilateral palsy may make it impossible for the patient to ingest any solid food; drink and semi-solids are as a rule more easily taken. There is always a danger in such cases that particles may enter the lungs and produce an aspiration-pneumonia.

Bilateral palsy is generally due to bulbar disease, and especially to the chronic nuclear degeneration that is a part of bulbar paralysis. The nerves of both sides may also be injured by meningitis or malignant growths at the base of the skull; they are often involved in diphtheritic neuritis. If the cause cannot be removed, the treatment must be symptomatic; the nasal or œsophageal tube should be used when the patient cannot take sufficient nourishment, or when there is danger of food entering the larynx.

Pharyngeal spasm is generally hysterical; it is intermittent, and usually dependent on external circumstances, as company that excites emotion or suggests sympathy. It is also a prominent symptom in tetanus and hydrophobia.

GORDON HOLMES.

### SWEAT-GLANDS, AFFECTIONS OF.

**Hyperidrosis**, excessive sweating, is a term that may be applied to the general sweating of fevers, but is better restricted to local forms, such as those occurring in the axillæ, or on the palms and soles. General abnormal states such as alcoholism, neurasthenia, and obesity, or the taking of arsenic, are important causal factors. Unusual types of regional sweating are also met with in certain diseases of the nervous system. When the condition is limited to the palms or soles, a very considerable degree of discomfort may be experienced. Not only are the affected parts constantly moist or even wet, but maceration of the skin takes place with accompanying liability to secondary infection and dermatitis. In hyperidrosis of the palms and soles this is so common as almost to constitute a part of the affection. The type

## SWEAT-GLANDS, AFFECTIONS OF

of hyperidrosis affecting the axillæ is relatively common in women. In **treatment**, consideration should first be given to any general abnormal state, such as obesity or alcoholism. Local treatment comes next, dusting powders or astringent lotions being employed. A suitable powder is salicylic acid 15 gr., boric acid 30 gr., starch 1 oz.; a lotion, tannic acid or alum 2 per cent. in watery solution. The administration of belladonna or atropine in therapeutic doses has been recommended, but can only be a temporary measure. Crocker's plan of giving a teaspoonful of precipitated sulphur in milk twice daily is sometimes successful but is apt to cause purging. Maceration and infection of the skin is best treated by baths of a mildly antiseptic type, such as the boric-acid bath, followed by the application of a weak antiseptic ointment—for example, ammoniated mercury 1 per cent., or ichthylol 4 per cent., in zinc ointment. Flat-foot when present should be rectified. Hyperidrosis of the axillæ is diminished and sometimes cured by irradiation with X-rays, a full pastille dose being given, suitably screened, and repeated if necessary after a sufficient interval. Temporary relief may be obtained by the application of a sponge previously wrung out in very hot water.

In **bromidrosis** the sweat becomes very offensive. This condition is usually limited to the feet, but occasionally may affect the axillæ or the perineum. There is usually accompanying hyperidrosis, the boots and stockings thus becoming fouled, and the skin macerated and infected. **Treatment** demands the most scrupulous cleanliness; the stockings should be changed frequently and the feet washed night and morning. If before being worn the stockings are soaked in saturated solution of boric acid, and allowed to dry, it will be found that the offensive odour is considerably diminished. The condition usually demands foot-baths, of boric acid in mild, and of potassium permanganate 1 in 1,000 in severe cases. Friction with alcohol is often useful, especially when the feet are cold and congested. The methods recommended for hyperidrosis should also be employed.

**Anidrosis** is a morbid state in which the secretion of sweat is greatly diminished or is absent. It may occur as a part of some other disease—for example, diabetes or ichthyosis—or it may be congenital, due to some developmental defect. When Anidrosis is not merely a symptom, Turkish baths, massage, radiant

## SYCOSIS, COCCOGENIC

heat, or other measures designed to encourage sweating may be employed.

Under certain conditions a coccal infection of the sweat apparatus takes place: to this the name **hidrosadenitis** is given. This infection occurs in the axillary region, giving rise to globular swellings the size of a pea or a marble. Spontaneous absorption may take place, or pus may form. The condition resembles and is often mistaken for furunculosis, and treatment is the same as for that affection.

In fevers, and particularly in acute rheumatism, tiny, very superficial vesicles may develop as the result of a process of splitting of the horny layer of the epidermis. This affection, to which the name **sudamina** is given, is of no importance and demands no special treatment.

Besides the above diseases, there are certain rare disorders of the sweat apparatus of which brief mention may be made. In **hidrocystoma**, tense, deeply seated vesicles arise on the face. Women of 40 years and upwards are affected. The eruption is persistent, but causes little or no inconvenience. **Treatment** consists in puncture of the vesicles, the clear or opaline fluid being allowed to escape. The name **granulosis rubra nasi** is applied to a form of hyperidrosis limited ordinarily to the tip of the nose, which becomes red, papulated, and beaded with tiny droplets of clear fluid. The condition begins in early life and continues for many years uninfluenced by treatment. It may disappear spontaneously.

H. MACCORMAC.

**SYCOSIS, COCCOGENIC.**—A chronic pustular folliculitis and perifolliculitis of the beard and moustache, due to infection by staphylococci.

**Etiology and pathology.**—The disease occurs in adult males, especially in those who are shaved by barbers. An analogous condition may affect the eyebrows and eyelashes and scalp or other hairy parts in both sexes. The disease generally begins as an impetigo, and infection may be due to the barber's brush or be set up by a purulent discharge from the nose or ear or a suppurative lesion in some other part of the body. When the upper lip is affected the cause is generally a chronic rhinitis. *Staphylococcus aureus* and in some cases *Staphylococcus albus* can be cultivated from the lesions. These organisms gain access to the hair-follicles, in which they set up suppurative inflammation, with the formation

## SYCOSIS, COCCOGENIC

of a small pustule around the hair. The hair itself is not destroyed and can be easily extracted, showing a swollen gelatinous root-sheath. Papules and nodules are interspersed with the pustules, and there is often considerable interfollicular inflammation.

**Diagnosis.**—The diseases most likely to be mistaken for sycosis are ringworm of the beard, impetigo, and eczema. *Ringworm* tends to form circular patches or rings in the early stages; short, greyish broken hairs can usually be detected, and these will show under the microscope the characteristic mycelial chains within the hair; sometimes thick nodular kerion masses are formed. *Impetigo* is more superficial, showing large yellow crusts not confined to the follicles, and will probably have a short history. *Eczema* is characterized by vesicles and serous crusts affecting the interfollicular skin and coalescing to form large areas extending beyond the hairy parts. There is a form of sycosis termed "lupoid" which spreads slowly at the margin and completely destroys the hair-follicles, leaving a superficial scar; this must be distinguished from *sypilis*, in which there is more infiltration and ulceration beneath the crusts, and from *lupus* by the presence in this disease of the so-called apple-jelly nodules.

**Prognosis and treatment.**—Sycosis is one of the most refractory of skin diseases, and sometimes persists for many years. Epilation by forceps has now been superseded by the X-rays, but although an immense amount of benefit may result from this method, the disease has a great tendency to relapse, since some cocci are left behind in the follicles and it may be necessary to repeat the epilation process until the hair-follicles are completely destroyed, with a risk of producing atrophy and telangiectasis of the skin. A preliminary epilation by X-rays, and even an application short of that required to produce epilation, will considerably assist in the removal of the suppurative folliculitis, and the treatment can then be continued with the application of mild antiseptic lotions and ointments such as a weak perchloride of mercury lotion and dilute nitrate of mercury ointment. An attempt may be made to raise the resistance of the patient's tissues to the staphylococcus by the administration of a vaccine—preferably autogenous, beginning with doses of about 100 millions repeated every ten days, and increased to 1,000 millions according to the response. This treatment is seldom curative, but is a useful

## SYNCOPE

adjunct to other measures, and may be combined with X-ray treatment. Intramuscular injections of colloidal manganese and stannoxyl by the mouth also help to control the pustulation.

S. E. DORE.

**SYOOSIS, TINEA** (*see* RINGWORM)

**SYMBLEPHARON** (*see* EYELIDS, AFFECTIONS OF).

**SYMMETRICAL GANGRENE** (*see* RAYNAUD'S DISEASE).

**SYMPATHETIC NERVOUS SYSTEM** (*see* VEGETATIVE [SYMPATHETIC AND PARASYMPATHETIC] SYSTEM).

**SYMPATHETIC OPHTHALMIA** (*see* UVEAL TRACT, AFFECTIONS OF).

**SYNCHYSIS SCINTILLANS** (*see* VITREOUS, AFFECTIONS OF).

**SYNCOPE. Etiology.**—Syncopal attacks are the direct result of an inadequate supply of blood to the brain. The outcome of such a condition is faintness, or even complete loss of consciousness, with the result that the patient collapses and sinks to the floor. The horizontal position restores the blood supply to the brain, and consciousness gradually returns.

It is not unusual to find that several members of the same family are afflicted with a liability to faint. They all possess an unstable vasomotor system, and not uncommonly this liability has existed since childhood.

Some persons feel faint on suddenly rising from a recumbent posture: their impaired vascular tone fails to meet the sudden demand made upon it, the requisite vaso-constriction in the limbs and abdomen is sluggish, and the blood collects in these areas, with a resultant drop in blood-pressure; the brain is imperfectly supplied, and a feeling of faintness or actual syncope ensues.

The same tendency is seen after a long illness. During convalescence the vaso-motor tone is only gradually regained. Owing to the same cause, even in the apparently healthy, the upright position, especially when the body is motionless, is sometimes followed by faintness. This happens when, for example, ladies are having clothes fitted on at the tailor's.

A mental shock or emotional disturbance, the sight of blood, a disagreeable smell, or other stimuli of comparable nature may cause syncopal attacks, and the slight degree of discomfort and mental anxiety associated with



## SYNCOPE

being vaccinated or receiving a hypodermic injection may be an adequate stimulus. Severe pain, as in colic or angina, is sometimes followed by faintness; in such cases it is probable that vagal stimulation is the dominant factor: the heart acts inadequately, the rate is diminished and the blood-pressure fails.

Probably the loss of consciousness associated with surgical shock is of a different nature. In such cases there appears to be stagnation in the capillaries, and only a fraction of the blood in the body is actually in currency. In these cases of surgical shock the abdominal veins are not engorged.

Syncope is sometimes associated with a diseased or degenerated myocardium, and patients suffering from advanced arteriosclerosis or aortic disease are liable to temporary loss of consciousness.

Syncope may be due to hæmorrhage, and sometimes is the chief signal pointing to internal bleeding, as in cases of peptic ulcer. Syncopal attacks are also common in Addison's anæmia, where the blood-pressure is extremely low. They are sometimes due to excessive smoking.

Recurrent syncopal attacks occur in Stokes-Adams disease. Such patients are liable to temporary cessation of ventricular systole. When the ventricles remain in diastole for a longer period than ten or twelve seconds, consciousness is lost, to be regained when the ventricles resume their activity.

In attacks of auricular flutter and paroxysmal tachycardia the ventricular rate may be so rapid and ineffectual that the brain does not receive a sufficiency of blood, and syncope results. A similar condition sometimes ensues during a paroxysm of auricular fibrillation. Fainting from these conditions is fortunately rare.

**Symptoms.**—Everyone is familiar with the symptoms and the outward appearances of a syncopal attack. A sense of limpness and giddiness, with nausea, is often the first indication. The colour leaves the patient's face and is succeeded by a deathly pallor of greenish hue, and a clammy sweat appears on the skin. If the condition is recognized at this point and adequate measures are taken, recovery may be rapid, otherwise faintness may pass into unconsciousness. The body is flaccid, the pulse slow and almost imperceptible, and the respirations may be shallow and sighing. Gradually the pulse returns, and with it consciousness. Vomiting and diarrhoea are occasional features in the recovery, and are

## SYNOVITIS

suggestive of a vagal origin. For some hours, or even days, the patient feels limp and useless and without energy; the return to normal is gradual.

**Treatment.**—When the early signs of approaching syncope are observed the patient should lower his head, tie his boot-lace, or perform some other action necessitating bending. In this way the large venous cistern in the abdomen is pressed upon, and blood is forced into the right side of the heart, and so into the arteries. If this is not sufficient to restore cerebral circulation the patient must be laid flat on a couch, pressure applied to the abdomen, and the feet placed on a higher level than the head. Smelling salts to the nose and friction of the hands and præcordium are also aids to recovery.

As consciousness returns, the face regains its colour with the improvement in the force of the heart, and the pulse becomes more normal; but it is not wise for the patient to sit up too soon after an attack. The complete recovery of the vaso-motor tone is gradual, and another attack of syncope is easily induced.

When pain is the cause of syncope, morphia must be exhibited.

For those who are liable to faint, general measures should be taken to improve the vaso-motor tone, such as a cold sponge-down in the morning, the daily performance of exercises in which rubbing of the skin plays a part, or which tend to increase the muscular tone of the abdominal wall, and participation in games played in the open air.

If anæmia is present, a prolonged course of iron and arsenic, Easton's syrup or other hæmatinic is indicated.

When some definitely abnormal cardiac rhythm, such as paroxysmal tachycardia, is the cause of syncopal attacks, appropriate treatment is indicated.

JOHN HAY.

**SYNOVITIS.**—Inflammation of the synovial membrane lining a joint.

**Etiology.**—Synovitis may be (a) traumatic, due either to direct injury or to a sprain or sudden wrench, (b) bacterial, or (c) toxic.

Many bacteria or their toxins may cause synovitis. Thus it may be due to the gonococcus, the spirochæte of syphilis, the tubercle bacillus, the toxins or bacilli of dysentery, of rheumatism or rheumatoid arthritis, etc. Septic organisms usually cause suppuration, but sepsis of the end of a bone may set up a serous synovitis of the adjacent joint.

## SYNOVITIS

**Pathology.**—The synovial membrane is inflamed, and a clear yellow fluid accumulates in the joint. In long-standing cases the ligaments may become relaxed. Lymph is sometimes deposited, and adhesions may form.

**Symptomatology.**—There is a swelling of the joint corresponding to the extent of the synovial cavity. When the amount of fluid is considerable, fluctuation may be obtained, swelling of the joint corresponding to the extent of the synovial cavity. When the amount of fluid is considerable, fluctuation may be obtained, and in the knee-joint the patella may be "tapped" against the femur. Pain is severe in acute cases, but in chronic synovitis is almost absent. In acute cases the movements of the joint are impaired, in chronic they are often quite free. Aspiration under antiseptic precautions proves the presence of a clear effusion.

**Diagnosis** of the presence of synovitis is easy, but, in the absence of traumatism, its cause is often difficult to determine, though by a process of exclusion this is usually possible. An X-ray photograph should be taken to detect fracture or disease of bone, the Wassermann reaction carried out, and one or other of the tuberculin tests performed, while the urethra, the buccal cavity and accessory sinuses, and any other possible focus should be examined for sepsis.

**Treatment. Traumatic synovitis.**—The immediate indications are to relieve pain, prevent and subdue swelling, and determine whether any more serious lesion is present. For the last purpose a radiogram is necessary. Pain is relieved by resting the limb in a position which is most comfortable to the patient. In most cases partial flexion of the joint is the best attitude, and may be maintained by a suitable splint. Evaporating lotions or fomentations may sometimes alleviate pain. If necessary, analgesics (aspirin, phenacetin, etc.) may be administered. Swelling is prevented or diminished by firm pressure of a semi-elastic (e.g. domett) bandage applied evenly over wool or gamgee tissue around the joint. The wool and bandage should be removed at least once each day and gentle massage performed. After three or four days, or when the swelling has subsided, very gentle active movements of the joint may be permitted. The massage and movements must be restricted or stopped if pain or swelling follows. It is inadvisable to immobilize the joint with splints for more than a few days. If the effusion distends the joint so as to cause great pain, aspiration

## SYPHILIS

with strict antiseptic precautions will cause relief.

**Bacterial and toxic synovitis.**—Here the main indication is to treat the cause, whether it be rheumatism, syphilis, gout, gonorrhœa, tubercle, sepsis, or any other disease. Local treatment consists in rest maintained by splints or by bandaging to a pillow, in applying fomentations for pain, and in aspirating the fluid if necessary.

**Chronic synovitis.**—Any form of synovitis may become chronic. The main principles of treatment are to apply firm pressure by wool and bandage or by strapping, to cause counter-irritation by applying Scott's dressing or by painting with tincture of iodine, to aspirate the fluid when necessary, to prevent adhesions by massage and passive movements, and to avert renewal of infection by treating the cause. Operation is necessary if the cause is a mechanical one, e.g. a loose cartilage, which so frequently causes synovitis of the knee.

ZACHARY COPE.

**SYPHILIS.**—A specific disease due to entry of a micro-organism (*Spironema pallidum*) into the tissues, either by inoculation into the skin or mucous membrane (acquired syphilis) or by transmission in utero (congenital syphilis).

### ACQUIRED SYPHILIS

In acquired syphilis a primary sore develops at the site of inoculation and is followed in a few weeks by lesions of the skin, mucous membranes, subcutaneous tissues, muscles, bones, viscera and central nervous system, which, at first generalized, and individually small (secondary stage), recur again and again at varying intervals throughout the patient's life. Each recurrence tends to be more and more localized and the lesions composing it to be larger and more deeply embedded. After many years, degeneration of the parenchyma of the brain (general paresis) or of the spinal cord (tabes dorsalis) may develop; these remote effects of syphilis are considered in CEREBRO-SPINAL SYPHILIS and other articles. From an early stage the blood-serum shows a peculiar property (Wassermann reaction) which persists until the disease is eradicated. Any or all manifestations after the primary may be omitted, and very occasionally no primary lesion appears. In congenital syphilis the systemic disease is the first manifestation.

**Etiology.**—The specific micro-organism was discovered by Schaudinn in 1905 and named by

## SYPHILIS

him *Treponema pallidum*. Subsequently this was changed to *Spirochaeta pallida*, but more recently C. Dobell has advanced reasons, accepted by the Medical Research Council, for naming it *Spirochaeta pallidum*. *Sp. pallidum* is a minute corkscrew-like organism varying in length from  $5.24\ \mu$  (average  $8-10\ \mu$ ); its diameter is  $\frac{1}{4}\ \mu$ , the distance between individual coils is  $1\ \mu$ , and the depth of each coil is  $1\ \mu$ . Owing to its low refractivity it requires for its demonstration in fresh specimens a special form of sub-stage condenser (dark-ground illumination). This is the most practical method of demonstrating *Sp. pallidum*, and under it the organism appears as a bluish-white, clean-cut, very delicate spiral, which is very active in its own ground but slow in moving from place to place. It alternately contracts and expands its coils, bends into loops, or forms itself into a right angle. It stains with considerable difficulty, the most usual methods employed being a modification of the Romanowsky stain, such as the Leishman or the Giemsa, which stains it rose-pink, or some method in which silver nitrate is employed, such as Fontana's stain, by which it is stained black. By one or other of these methods it has been demonstrated in every syphilitic lesion, including the brains of general paralytics, and its etiological connexion with syphilis has been proved experimentally on animals by Metchnikoff and Roux and numerous other workers.

The organism was cultivated first by Noguchi, who showed that it is an anaerobe. It is purely a parasite, having a life of only a few hours under natural conditions outside the body, though, sealed under a cover-slip, I have seen a specimen retain its activity for as long as six weeks. It is killed at once by drying and by the application of much feeble antiseptics than suffice to destroy ordinary pathogenic organisms.

The usual method of transmission is by sexual intercourse. It does not seem necessary for the person transmitting the disease to be suffering at the time from syphilitic lesions of the external genitals. At least, this appears to be the case when men convey the infection, and it is clear that, in the earlier stages of the disease at any rate, the semen contains the virus. The period during which a person suffering from syphilis is liable to convey the disease by sexual intercourse varies; after the second year the chances diminish, and it is very unusual for infection to be passed on in this manner after the fifth year. There is no

doubt, however, that infection can be transmitted to the fetus by an infected mother up to a much later period, and cases have been cited in which syphilis was thus conveyed as long as eighteen years after infection of the mother. Accidental infection occurs by contamination of any minute abrasion with secretion from a syphilitic lesion. Those lesions which are richest in spirochaetes, such as the primary sore and the early secondaries, are the most dangerous from this point of view. As the age of the infection increases, it becomes increasingly difficult to discover *Sp. pallidum* in the lesions, and although it is possible to infect an animal with secretion from later or tertiary syphilides, the chances of conveying the disease at this period by ordinary social intercourse are extremely slight. Even in the earlier stages, in spite of the fact that contamination of an abrasion with the virus received direct from an early syphilitic lesion (teeming as these usually are with spirochaetes) almost invariably results in infection, the chances of accidental infection by ordinary social intercourse must be very slight; hence the very low proportion of extragenital chancres. The explanation probably lies in the low resistance of the virus to external conditions. These facts are often forgotten by many who appear to think that a syphilitic patient is rather more dangerous than one suffering from plague or smallpox, and that the room which he has inhabited must be disinfected with particular thoroughness.

**Invasion of the tissues.**—Although a number of days elapse before a visible lesion appears, within a very few days after infection histological examination of the tissues at the site of inoculation shows that changes are already commencing, and animal experiments have demonstrated that, by this time, the virus has spread throughout the body, infecting particularly the spleen, bone-marrow and testicles. The rapid spread of the spirochaetes to the deeper tissues is shown by the fact that, although it is a very delicate organism, the local application of antiseptics to the site of inoculation has succeeded in preventing infection of monkeys only up to eighteen hours after inoculation (see p. 312). No protective vaccine has been discovered, but Magian has succeeded in preventing syphilis after experimental inoculation of a man by injecting salvarsan intravenously.

**Pathology.**—The syphilitic lesion of every stage is a granuloma composed of an exudate





PLATE 34.—HARD CHANCRE.

## SYPHILIS

of plasma cells and lymphocytes around the parasitic focus. Regarding this exudate as the reaction of the tissues to the toxins evolved by the virus, we may explain the differing clinical appearances described below, which characterize the successive recrudescences of activity of the virus, by the increasing sensitiveness of the tissues. As we have seen, syphilis is a disease in which there are alternate periods of activity and quiescence. In an uncured case the periods of quiescence are probably only apparent, as is shown by the fact that the blood serum gives the syphilitic (Wassermann) reaction when no other signs of disease are apparent. It seems probable that, at these times, the virus is giving off enough poison to produce the reaction, but insufficient to cause the tissues to react grossly. The tissues are resisting, but the effect of the struggle on them is shown when the spirochaetes temporarily gain the advantage and, presumably, give off sufficient toxin to provoke a gross tissue reaction. The response of the tissues is the greater the older the infection—i.e. the longer the virus has been acting on them. Thus, if one were to obtain secretion from (i) a small head, (ii) a secondary papule when the infection was about three months old, and the same size or bigger, and (iii) a large tertiary lesion say ten years old, in the first specimen would be found a large number of the spirochaetes, in the second only a moderate number, and probably none would be found in the third. Early in the disease, then, a large number of organisms produce only a small lesion, a little later a moderate number produce a moderate lesion, and still later a very small number produce a very large lesion. Histologically, all the lesions are the same; the difference is in their size and their subsequent course.

An important feature of the action of *Sp. pallidum* is its effect on arteries. The organism has a predilection for the perivascular lymph spaces, and acts particularly on the vasa vasorum. The result is degeneration of the arterial coats and thrombosis of such small arteries as the vasa vasorum and vessels involved in syphilitic granulomata. In the case of larger arteries the middle coat becomes infiltrated and the internal undergoes compensatory thickening (endarteritis obliterans). The general effect depends on the artery, which may dilate with a resulting aneurysm, or may gradually become occluded. Illustrations of this are seen in aneurysm of the aorta and other large arteries, and in thrombosis of

cerebral, spinal and coronary arteries. In the case of the primary lesions the occlusion of arteries may result in failure of antisyphilitic remedies to reach the parasite in the depths of the lesion, and it is now a fairly common occurrence for these to reawaken months after all signs of the disease, even the blood reaction, have disappeared and to give rise to a lesion at the site of the primary sore which approaches in clinical characters that of a tertiary lesion (*recurrent chancre*). In tertiary lesions the cutting off of nutritional supplies to the centre leads to necrosis and liquefaction. This is seen in the clean-cut ulceration which characterizes tertiary syphilitic granulomata. If bone is involved in the tertiary gumma it may necrose throughout as in the flat bones, or only partially as in the long bones. Where necrosis does not occur the granulomatous tissue of a syphilitic lesion tends to become more and more fibrous, though at the same time natural forces work towards its removal. There is, so to speak, a race between the two processes. If the amount of exudate is comparatively small, as in some primary and in practically all secondary lesions, the exuded cells are practically all removed. If the exudate is a large one, a considerable proportion of it may reach the fibrous stage and become very resistant to absorption before the natural scavengers have had time to remove it. Thus many primary lesions are marked by scars, of cartilaginous hardness, for years afterwards, and gummata, their centres having liquefied, may be enclosed by dense connective tissue. Bone gummata, in fact, are often ringed by heaped-up bone of ivory hardness.

The changes which occur when the parenchyma of the central nervous system is invaded are described in CERE BRO - SPINAL SYPHILIS.

**Primary Sore and early course.**—The incubation period varies from a minimum of about ten to a maximum of about sixty days, with an average of four to five weeks, and is succeeded by the appearance of a small papule at the site of inoculation. This quietly enlarges to a round or oval sore about the size of a threepenny-bit (PLATE 34); the centre usually becomes eroded or perhaps more deeply ulcerated, and the broken surface is surrounded by a dull-red areola varying in width from  $\frac{1}{4}$  to 2 or 3 millimetres. A characteristic of great diagnostic importance is that, extending beyond the confines of the sore, the tissues are indurated and matted with the sore into a

## SYPHILIS

button of indiarubber-like consistence. With age this induration becomes more and more pronounced until, in the case of some primary sores, the feeling on palpation is as if there were a button embedded in the tissues. The sore does not bleed easily when scraped, but serum oozes freely from it and this serum usually teems with the spirochaetes of the disease. The sore is comparatively painless.

Those are the main characteristics of all primary syphilitic sores; the individual features vary in prominence with the site. Thus the most indurated are those on the under-surface and mouth of the prepuce, where apparently the process has an opportunity of extending deeply as well as superficially. The induration of a sore at the reflection of the prepuce on to the corona glandis results in a very characteristic appearance; when the prepuce is retracted the lesion flicks over like a plate turning on its edge. Induration is easy to elicit in sores affecting one wall of the fossa navicularis, which then feels as if a tiny, hard plate were embedded in it. In primary sores of the glans itself induration is difficult to elicit, owing to the tightness of the tissues, but the sore is easy to recognize by its dull-red areola, glistening surface, even contour, eroded centre, and indolent progress. Primary sores of the skin do not usually feel so definitely indurated as those of the under-surface of the prepuce, and are usually covered by a dark scab. They are readily recognized by their evenly-round or oval contour, the depth to which the underlying tissues are matted, and their indolence. Ulceration is usually a more prominent feature of sores affecting the under-surface of the prepuce than elsewhere, save, perhaps, the lip and tonsil. Almost all primary sores are comparatively painless, but when affecting the terminal phalanx of a finger or thumb they may give rise to excruciating pain; this, no doubt, partly accounts for the fact that primary sores in this situation are so often diagnosed as whitlow. The primary sore affecting the prepuce, the skin of the penis, or (especially) the female labia may be accompanied by a toughly indurated oedema of the affected parts by which it may be entirely obscured; the colour is somewhat livid.

The course of the primary sore varies greatly. In some cases the lesion is fleeting and apt to pass unnoticed. The history of a substantial proportion of cases of tabes and general paresis is that the initial lesion was either unnoticed or was very trivial. Apart

from the neglect of treatment to which this may give rise, it is possible that the virus which enters without causing a great local reaction may thereby gain more easily a footing in the parenchyma of the central nervous system. The ordinary sore which remains untreated lasts for a month or longer, and long after the erosion has healed over, a button of indurated tissue may remain to mark the site. Weeks or months later the sore may break down again, the resulting lesion approaching then more closely to the characters of a tertiary ulcer. It is probable that in these cases the spirochaetes buried in the depths of a mass of granulomatous tissue composing the lesion have gradually re-awakened to activity. When a sore becomes infected by secondary organisms, ulceration is a more prominent feature, and in rare instances the ulceration is phagedenic. In such cases the surrounding tissues very rapidly become black and necrotic, and the resulting loss of tissue may involve large portions of the external genitals.

Shortly after the appearance of the primary sore the nearest lymph-glands often become painlessly enlarged, and in the case of the penis the lymphatics running from the sore can frequently be felt below the skin. The affected glands may reach a large size, bulging out the overlying skin, and this, with the fact that there is no reddening of the skin or other sign of acute inflammation, is often sufficient to give the clue to the nature of the sore on which it depends. Syphilitic buboes do not usually suppurate, but may do so if the sore has become contaminated by secondary organisms, so that suppuration should not weigh heavily against the diagnosis of syphilis. A week or so after the local lymph-glands have begun to enlarge there is universal adenitis, which can be appreciated by palpation, particularly of the epitrochlear, axillary, and cervical glands. About this time, or when the sore is about fifteen days old, the blood serum usually gives a positive Wassermann reaction. The percentage of cases in which this reaction is given increases with the age of the disease until the outbreak of the skin lesions which manifest the next or secondary stage. Practically 100 per cent. of patients in the secondary stage give a positive Wassermann reaction. Strictly speaking, the secondary or generalization stage of syphilis begins with the first appearance of a positive Wassermann reaction, but it is still usual to date it from the first appearance of generalized skin lesions.

## SYPHILIS

**Diagnosis of the primary lesion.**—The guiding characteristics by which primary syphilitic sores are distinguished from others are the incubation period, colour, indolence, comparative painlessness, slighter tendency to bleed, indolent enlargement of neighbouring glands, and the presence of *Sp. pallidum* in the serum that oozes from the sore. The length of incubation period is a guide only when the patient has not been exposed to infection for over ten days.

Of sores which may appear on the genitals, *herpes* may occur at any period and so appear to have a long incubation period, but the individual herpetic sore is pin-head in size and very superficial, looking as if the top quarter millimetre of tissue had been clearly punched out. Usually herpetic lesions are multiple and fairly closely set, so that, when the tops of the minute blisters have been rubbed off, a composite lesion is seen made up of a number of minute circles or of segments of circles. Herpes is not accompanied by induration or enlargement of neighbouring glands, and is usually attended by some degree of irritation.

*Chancroid* has an incubation period of only a few days; the sore is of a more inflammatory nature, being undermined and more definitely ulcerated, with a sloughing or a "worm-eaten" base. The edge is merely tipped with red, and that of a shade which is much brighter than the areola surrounding a primary syphilitic sore. It should always be remembered, however, that a sore may begin as a chancroid and take on syphilitic characters afterwards, owing to mixed infection and the different incubation periods of the two types of sore. It may thus happen that a case of mixed infection is dismissed at once as chancroid owing to its having the characteristics mentioned above and the failure to find *Sp. pallidum* in the serum exudate; the thickening and induration which develop later are apt to pass unnoticed, and the first event denoting syphilis is the outbreak of a skin eruption. A good rule is to continue microscopical examinations at intervals until the sore heals and to test the blood for the Wassermann reaction for three months. Chancroid may be accompanied by a bubo which is more inflammatory in type than the syphilitic, and tends to suppurate. A syphilitic bubo may, however, suppurate, so that it would be a mistake to exclude syphilis on the sole ground of suppuration. One would say rather that if the glands, though distinctly enlarged, showed no signs of active inflammation, it would

be strong evidence in favour of their being syphilitic.

*Scabietic runs* on the glans and skin of the penis are often diagnosed as syphilis. They are mound-like, not eroded, have no areola, are not indurated, and, of course, the serum exudate contains no syphilitic organisms.

Syphilitic sores in parts of the body other than the genitals are often overlooked, mainly because syphilis is not thought of in those parts. A unilateral tonsillitis should arouse suspicion, especially if associated with painless enlargement of the submaxillary glands on one side. Similarly, the clue to the nature of a lip chancre may be given by the glands. Primary sores affecting the terminal phalanx of a thumb or finger are often extremely painful and simulate whitlows rather closely. The syphilitic sore is more brawny, and remains so considerably longer after the sore has been lanced.

In any case of doubt a specimen of serum should be taken from the sore and examined microscopically. This should be done before any antiseptics have been applied, as they kill off spirochaetes in the superficial layers and prejudice the success of the examination. To obtain a specimen which has to be sent away to a laboratory for examination, the sore should first be cleaned with a swab of lint moistened with saline, and its margins scraped with the edge of a scalpel or a vaccination lancet. This results in serum oozing from the scarified area, especially when the sore is squeezed. The serum, which should contain only a slight amount of blood, is allowed to run into a capillary tube (like a vaccine-lymph tube) both ends of which are sealed off afterwards with sealing wax or by melting in the flame. If antiseptics have been applied the specimen should be obtained from the deeper layers by puncturing the margin of the sore with the point of the instrument, squeezing, and waiting for the bleeding to stop before collecting. Alternatively, a good method, which should always be adopted if the sore looks at all suspicious and the organism has not been found in the specimens taken directly from it, is to puncture the nearest enlarged gland and aspirate a little of the gland juice. A moderately stout needle is run obliquely into the gland, and a few minims of sterile saline injected into it. The gland is massaged and aspiration applied by a syringe. The fluid aspirated is forced into a watch-glass and collected in a capillary tube as above. It is always a good plan to send two or three speci-



## SYPHILIS

mens, as it often happens that the first specimen is negative, while the second, taken some minutes later, when serum has had an opportunity of oozing up from the deeper layers, is positive. When the necessary apparatus is at hand it is always better to examine the specimen at once.

*The dark-ground illumination.*—This, the best method of examination, is carried out as follows:—

1. The drop of serum is collected on a cover-slip, which is lowered, specimen downwards, on to a slide of special thickness as recommended by the maker of the dark-ground condenser described below. The specimen is made very thin by pressing it, cover-slip downwards, on filter paper.

2. In place of the ordinary sub-stage condenser, a special, dark-ground condenser is fitted. There are various patterns of dark-ground condenser, which may broadly be divided into those with hemispherical and those with paraboloidal reflecting surfaces, but both are fitted and worked on the same plan. After it has been fitted, the condenser is racked up until it is flush with the stage and is centred as follows: By means of two centring screws the microscopist brings into the centre of the field two concentric circles engraved on the upper surface of the condenser, which he watches through a  $\frac{3}{8}$ -in. objective and a low eyepiece.

3. A drop of cedar oil is placed on the top of the condenser and another on the under-surface of the microscope slide. The latter is then lowered on to the stage so that the two drops coalesce to form a film between the slide and the condenser. This film should contain no air-bubbles.

4. The light of an arc lamp is condensed through a bull's-eye lens on to the plane mirror of the microscope, and this is so directed as to illuminate the specimen.

5. The specimen is viewed through a  $\frac{3}{8}$ -in. objective and a low eyepiece, and, while it remains in focus, the condenser is racked so as to make as bright and small as possible the central bright area which is obtained.

6. A special stop, supplied by the makers for the purpose, is fitted into the  $\frac{1}{2}$ -in. oil-immersion objective, and the specimen is then viewed with this and a No. 4 eyepiece.

Spiral organisms other than *Sp. pallidum* may be seen in a specimen obtained from the genitals or the mouth. They are largely eliminated by taking care to clean the surface of the lesion before collecting the specimen, but some may still be included. All coarsely spiral organisms which appear thick should be excluded at once. To the observer who has once observed *Sp. pallidum* closely only three others will cause difficulty:—

1. A fairly fine spirochæte with closely set spirals often found in specimens from the geni-

tals. It is about twice as thick as *Sp. pallidum*, shines more brightly, has a slightly rusty tinge, and its spirals are by no means so cleanly cut as those of *Sp. pallidum*.

2, 3. Two very fine spirochætes found in the mouth, and both as fine as *Sp. pallidum*. One is distinguished by its curves being angular rather than round, the other by its spirals being much more closely set, so that it looks like a piece of twisted silk.

Films of secretion dried on a microscope slide may be stained as follows:—

### A. Leishman's Stain

Leishman's stain in powder	. . . 0.15
Methyl-alcohol, acetone-free	. . . 100

Grind the stain to a fine powder in a clean mortar; add a few drops of the alcohol and grind to a paste; slowly add more alcohol, whilst grinding, until about 15 c.c. have been added. Allow to stand for a few minutes, to allow the undissolved particles to settle to the bottom; decant into a clean bottle and repeat the process with the remainder of the solid stain in the mortar. Continue until all the powder and stain have been transferred to the bottle. It is best to keep the prepared stain for three days, with frequent shaking before using it.

The slide on which the film is to be made must be perfectly clean and free from grease. The film must be as thin as it is possible to make it. The material may be lightly rubbed over the surface of the slide or, if of fluid consistency, may be spread by any of the means employed in the preparation of blood-films. The stain and water, in the usual proportions of two parts of water to one of stain (which will be found to be contained in about the same number of drops), are mixed beforehand in a watch-glass, and poured directly on to the unfixed film. Optimum staining takes place in about twenty-five minutes, and the only additional precautions to be observed are that the distilled water used for washing off the stain must be employed with great gentleness, and that the blotting with cigarette paper must be done by slight pressure and without rubbing. By this procedure the red cells are, of course, de hæmoglobinized and, in addition, a great deal of detritus is dissolved off from the film; the leucocytes, tissue-cells and bacteria remain.

This method has the advantage that the greater freedom of the film from debris and extraneous matter makes the detection of *Sp. pallidum* easier.

### B. Tribondeau's Modification of Fontana's Method

1. A thin film of the exudate, as free from blood as possible, is spread on a slide and allowed to dry. It is covered repeatedly for 1–5 minutes with the following (Ruge's) solution:



PLATE 35.—MACULO-PAPULAR SYPHILIDE.



## SYPHILIS

Pure acetic acid . . .	1
Formalin, 40 per cent. . .	2
Distilled water . . .	100

Alcohol is dropped on the slide and then flamed.

2. The following mordant is then applied :

Tannin (alcoholic or ethereal) .	5
Hot distilled water . . .	100

- This is warmed gently till steam rises, and then allowed to act for thirty seconds longer.

3. The specimen is washed under the tap for a few seconds, the excess water thrown off, and the slide covered with Fontana's solution, which is made as follows : To a 5-per-cent. solution of silver nitrate, ammonia is added drop by drop with a capillary pipette until the sepia precipitate, which forms at first, disappears. To this solution is added more silver-nitrate solution until a solution is left which remains slightly cloudy on shaking.

The slide covered with this solution is warmed gently till steam rises, and the solution is then allowed to act for thirty seconds longer.

The spirochaemes of syphilis appear dark brown to black, and are easier to find than in specimens stained by other methods. They are slightly thicker, and this must be allowed for in coming to a decision as to their nature.

Another method of demonstrating *Sp. pallidum* is to stain the background as follows :

### C. Collargol Method

A solution of collargol is made by placing 1 part in a bottle with 19 parts of distilled water and shaking energetically.

A drop of the exudate from the suspected lesion is placed at one end of a clean slide, and a drop of the collargol solution next to it. With the help of a platinum loop the two are mixed, and the mixture is spread with the end of another slide as in making a blood-film. The organisms appear white on a brown-black background.

One other method remains to be described :

D. Benian's Modification of Burri's Method  
Prepare the following :

(a) Congo red . . .	2
Distilled water . . .	to 100
Filter before use.	
(b) Hydrochloric acid . . .	1
Absolute alcohol . . .	to 100

On a perfectly clean slide place a drop of the exudate to be examined, and a drop of solution (a) alongside it. Mix the two with the help of a platinum loop, and spread fairly thickly like a blood-film. Allow to dry, and then flood the slide with solution (b), which is allowed to act for one minute.

The background is blue, and the organisms stand out from it as white corkscrews.

In a proportion of cases the only manifestations of syphilis are the primary sore and a positive Wassermann reaction of the blood-serum, no further obvious sign appearing throughout a long life. In others no general skin lesions appear, but years later one or more gummata develop. In still others the primary sore is inconspicuous, and no further signs appear until, many years later, the patient develops tabes or general paresis.

**Secondary stage.**—In the great majority of cases the first evidence of what is called the secondary stage appears three or four weeks after the sore, as a generalized rash and characteristic mouth lesions. The first eruption usually starts on the sides of the trunk in the form of a pinkish **erythema**, the spots varying in size from a split pea to a little-finger nail, and following the folds of the skin. The eruption spreads gradually over the trunk and limbs, and the individual spots deepen in colour with age to a dull-red or somewhat brownish tint. At first they may be difficult to see, requiring a good light, but they usually become more manifest after the patient has stood stripped for a few minutes, when the blanching of the normal skin produced by the cold provides the necessary contrast. The erythematous rash fades in a few weeks, leaving little or no staining. It may recur at a later period, when it is much less generalized, while the spots are set in circles the size of a sixpence or larger.

After the fading of the first roseola a peculiar change in the distribution of the pigment may occur, especially in brunettes. The result is that the neck is generally discoloured, or, more commonly, assumes a dappled appearance (leuco-melanoderma) which is diagnostic of syphilis.

**Diagnosis of the early eruption.**—The maculo-roseolar syphilide is fairly easy to distinguish by the history of a primary sore with indolent adenitis; by the subcuticular, deeply-grounded appearance of the spots, which first appear on the flanks, very rarely itch, and are of a pinkish or dull-red rather than a bright-red tinge, and by the coincidence of a positive Wassermann reaction. Other erythemata are brighter red, more irritable, and often affect the backs of the hands. *Seborrhœa* is more superficial, not well grounded in the skin, and is more scaly, the scales being greasy. *Ringworm* is more superficial and irritable, and the fungus can be found in scrapings from its border. *Tinea cruris* or *dhobie's itch* affects a triangle at the upper and inner part of the

## SYPHILIS

thigh; it is brighter red, more irritable, and quite superficial. *Drug rashes* are more inflammatory and irritable; they appear more suddenly and are associated with a history of the patient having taken such a drug as copaiba, cubebs, antipyrin, quinine, or belladonna. The *eruptions of specific fevers* are usually accompanied by more pronounced constitutional symptoms. *Marbling of the skin due to cold* might be mistaken for a syphilide, but here the normal skin is framed by the abnormal, while in the maculo-roseolar eruption the macules are framed by normal skin. *Pityriasis rosea* is often mistaken for syphilis, but the lesions are brighter in colour and more irritable; they tend to become annular with their centres covered by branny scales.

**Later secondary eruption** (PLATES 35, 36, 37).—Following closely on the erythematous eruption is the **papular**, which takes a number of different forms. The commonest, and generally the earliest, consists of numbers of dome-shaped, dull-red, indurated papules distributed generally over the trunk, limbs, and face. Most of them are about the size of a lentil, but scattered among the smaller papules may be a considerable number of others which are larger and sometimes attain the size of a threepenny bit. Usually, the later the papular syphilide appears the greater is the proportion of large papules in it. Variations of the ordinary papular eruption are the *papulo-squamous*, *squamous*, *papulo-pustular*, and *pustular*. In the *papulo-squamous* a large proportion of the papules are covered at their centres by a few loose scales. The *squamous* syphilide is a papular eruption in which scaling is a still more prominent feature. In the *papulo-pustular* syphilide the centre of the papule necroses, and the appearance is rather that of a suppurating acne spot. An extension of this is the form in which the whole papule breaks down and the *pustular* syphilide results. When extensive the *pustular* syphilide may resemble a varicellar or a variolous eruption. A more severe and malignant form is that in which the papule breaks down quickly and the underlying tissues are eroded or ulcerated. As the destruction of tissue extends, the secretion dries to a crust. This may become heaped up by the laying down of successive layers, and the result is a blackish crust shaped like a limpet (*rupia*).

The papular syphilides take a different form in moist situations. Thus, between the buttocks and on the lateral surfaces of the scrotum, as

also on the labia, they become very prominent and wart-like and are covered with sodden epithelium, which gives them a greyish-white appearance; they are called *broad condylomata*. The whole of the contiguous surfaces of the buttocks may be covered with these lesions, which may diffuse into a large plaque with a few outlying growths. On the scrotum condylomata tend to be more prominent than between the buttocks, probably because the surface is not rubbed away so freely as in the latter situation. The papular syphilide assumes another very characteristic form on the scrotum. It is best displayed by making the patient stoop down and then pulling his scrotum out between his legs; a number of glistening-white, slightly raised rings with brownish centres are then seen. Between the toes, under the pendulant mamma, and in almost all moist situations, papules tend to run together and, their sodden covering often becoming rubbed off, a moist, pinkish-red surface fringed with loosened epithelium is left. Condylomata between the buttocks and on the scrotum and moist papules generally exude serum freely; the parts appear unnaturally moist, and the smell is often overpowering. The papular syphilide may be well marked on the forehead, following the margin of the hair, and it is often possible to find many papules in the hairy scalp. On other parts of the face, especially about the naso-labial fold and the chin, the papules are often set in rings. When the papular eruption of the type under discussion recurs on the body it tends to be distributed in rings, as in the case of other types of recurrent syphilide.

The *follicular* or conical type of papular syphilide usually occurs at a later period than the dome-shaped variety just described. There are two main varieties, the small and the large follicular. The *small* follicular syphilide occurs in clusters of minute, pin-head, brownish papules affecting the hair-follicles. The clusters are often diamond-shaped, and particularly thickly distributed on the back. The affected areas, each about 1 cm. across, have a goose-skin appearance, and the part generally looks as if it had not been properly washed. The *large* follicular syphilide is usually disposed in circles the size of a half-crown or larger, the centre of the circle being generally occupied by a particularly large papule, round which a ring of smaller satellites is ranged; this is the *corymbose* syphilide. It usually appears comparatively late, say about the end of a year or



PLATE 36.—PAPULAR SYPHILIDE.  
*(Ricketts and Byles.)*



## SYPHILIS

eighteen months, and its characters approach closely to those of the nodular cutaneous syphilide, to be discussed under tertiary lesions.

**Diagnosis of papular and pustular syphilides.**—The ordinary dome-shaped papular syphilide is usually easy to distinguish from a non-syphilitic eruption. Among strongly guiding features are the history of a sore, a positive Wassermann reaction, and presence of *Sp. pallidum* in the serum which oozes from the spots when they are scarified. The microscopic test should always be applied in any doubtful case, and very rarely fails, even with the papulo-pustular or the pustular syphilide. The indurated feel of the papule, its readiness to scale, and its dull or pinkish-red colour are valuable diagnostic signs, as is also the association with mouth and throat lesions. Altogether, the ordinary papular syphilide could hardly be mistaken for a non-specific eruption. *Acne spots* are more inflammatory, and tend also to affect the upper front of the chest and between the shoulders behind, rather than the flanks, loins, and limbs. *Molluscum spots* are white and umbilicated, while caseous matter can be squeezed from their centres. *Lichen ruber planus* is characterized by flatter, smaller, polygonal spots of a violet tinge and waxy covering; it is more irritable and favours the nape of the neck. A papulo-squamous or squamous syphilide may suggest *psoriasis*, which is usually less indurated, more superficial, bleeds at a number of points when lightly scraped, and affects the extensor rather than the flexor surfaces of the limbs; the scales are more silvery, and in moist situations the rash remains true to type, contrasting with the syphilide, which here becomes sodden with the secretion that freely oozes from it. The well-marked pustular syphilide may resemble varicella or variola, but is not attended by such constitutional disturbance. *Varicellar spots* are more superficial and not associated with mouth and throat lesions. *Variola* is quicker in development and change, and tends to affect the backs of the hands and wrists. *Bromide and iodide eruptions* appear more suddenly and are considerably more irritable. The deeper forms of pustular syphilide with considerable crusting, such as the superficial and deep ecchymatous, or the rupial, are distinguished from ordinary *impetigo* by the darker colour of the crusts, the circular rather than linear shape of the lesions, and the greater degree of tissue destruction below the crusts. *Scabies* is often mistaken for a crusted syphilide, and perhaps

more frequently the contrary occurs. The individual scabietic lesion is easy to recognize, but the tendency is to forget that scabies and syphilis often coexist.

The syphilitic **eruption in the mouth** is similar in character to the papular eruption in other moist situations of the body. On the mucous surface of the lips and the pillars of the fauces the syphilide is a greyish-white patch edged with a pinkish-red areola which marks it off from the surrounding mucous surface. The mucous patch on the lip is usually round or oval and, if it crosses the angle of the mouth, is fissured. On the pillars of the fauces the appearance is that of a snail-track creeping up over the pillar, perhaps on to the soft palate. On the sides of the tongue fissuring and ulceration are more pronounced, but on the under-surface the lesion may be more condylomatous in type. The secretion from these lesions teems with spirochaemes and is very contagious. Secondary syphilitic mouth lesions are easily diagnosed by the characters mentioned above.

The **hair** is shed to a varying degree in the secondary stage. In most cases there is some thinning, which does not produce a very noticeable result; in others it falls in a patchy manner, giving the back of the head a moth-eaten appearance; while in still others the patient may become temporarily bald. The beard and eyebrows may participate in these changes. The **finger-nails** may show characteristic changes, more especially in the recurrent secondary stages. The end of the finger becomes pinkish-red and bulbous, and the reflection of the skin on the nail is occupied by weeping granulations. The nail becomes brittle and lustreless and is shed.

Joints and bursae are not often affected in secondary syphilis, but occasionally an **acute synovitis** occurs. It is fairly painful, and usually worse at night. A more indolent form of synovitis causes swelling without pain or great limitation of movement. The tendon-sheaths may be affected similarly, and the tendency to formation of adhesions may lead to permanent limitation of movement. **Mild periostitis** may occur in the secondary stage, but bone-affections are commoner in the tertiary.

From about the sixth month, or even earlier, the patient may develop symptoms pointing to syphilitic disease of the central nervous system, which are dealt with in CERE BRO-SPINAL SYPHILIS.

**Tertiary lesions.**—There is no sharply dividing line between the secondary stage and



## SYPHILIS

the tertiary, since the earliest tertiary lesion, the nodular cutaneous, is merely a collection of papules which are more deeply embedded and tend more to ulceration than the papules of what is called the early secondary stage. Since the pathology of all syphilitic lesions is qualitatively the same and the differences consist in degree only, this is not to be wondered at. With each recurrence of the activity of the spirochaetes the tissue reaction tends to be greater and greater, so that eventually, for an apparently very slight stimulus, as judged by the number of spirochaetes to be found in the late tertiary lesion, the tissue response is enormous.

The **nodular cutaneous syphilide** occurs in one or more isolated areas of the skin as a collection of skin gummata, each about the size of a pea, often running into one another to form a more or less continuous, brownish-red ridge which describes a circle or is made up of the segments of a number of circles so as to produce a snake-like line of varying length. Sometimes the affected area looks as if a number of groups of concentric circles had been described on the skin; sometimes two or three very short concentric arcs are seen. The affected area may be as small as a finger-nail or larger than a hand. The individual gummata may degenerate only so slightly as to produce some scaling, or may ulcerate more deeply and become crusted. The lesion extends centrifugally, and leaves in its wake a reddish stained area which shows little evidence of scarring; or a supple, papery scar which shows well the concentric distribution of the lesion. In some cases, where the ulcerative process is more intense, extension is more rapid than healing, and a large patch of small skin ulcers may be left in the wake of the advancing circles of new lesions. The areas of the body which are more commonly affected are about the iliac crests, over the shoulder-blades, the palms of the hands, the flexor surfaces of joints, and the nose, forehead, and mouth region. The areas chiefly affected are those exposed to injury or constant friction, and an odd distribution can usually be traced to the patient's occupation exposing the area to such stress or injury. A similar type of lesion may affect the soft palate and lead to considerable ulceration and deformity.

**Gummata of the tissues underlying the skin** are usually later than the skin gummata, although this is by no means an invariable rule. Those of the subcutaneous tissue and muscles grow up as lumps which vary in size from a

shrapnel bullet to an orange or larger. They are painless, quietly expanding growths which spread gradually to the skin on one side and to bone on the other. They tend to break down in the centre and, opening through the skin, discharge their gummy contents, leaving a clean-cut ulcer with overhanging edges and a tough, wash-leather slough occupying the base. Sometimes, instead of a discrete gumma forming in the muscles affected, they are diffusely infiltrated, and much deformity results when the degenerated tissue is replaced by scar.

The **joints, bursae, and tendon-sheaths** are not often invaded in tertiary syphilis. When they are, the infected parts are, again, those most exposed to stress and strain, such as the knee-joint and the prepatellar bursa. The swelling follows the shape of the affected membrane, and its nature is easy to recognize by its soft, indiarubber-like consistence, as also by the fact that it follows the lines of the joint, bursa, or tendon-sheath affected, and by the absence of signs of active inflammation.

The **bones** are affected in tertiary syphilis in different ways, depending largely on whether the process starts in the periosteum or in the bone itself. In the long bones the most usual manifestation in acquired syphilis is a localized gumma which eventually necroses in the centre while the edge becomes converted to a raised circle of ivory-hard bone. The contents are discharged through the skin, leaving an intractable ulcer, at the bottom of which bare bone may be felt. If the process begins deeper the local swelling may not be so obvious, and eventually, with discharge of the necrotic contents, one or two fistulous openings lead to the interior of the bone. Diffuse osteitis and periostitis are not so common in acquired as in congenital syphilis. They cause general enlargement of the bone, and a sign of congenital syphilis in adolescents and adults is the sabre-scarred tibia resulting from diffuse periostitis in earlier years. The bone is thickened from before backwards, and its anterior crest is curved with its convexity forwards, giving the bone the shape from which the condition derives its name. Of the long bones, the most commonly affected in acquired syphilis are the clavicle (sterno-clavicular joint), sternum, ribs, tibia, and femur; but no bone is immune, and the process is particularly apt to affect those which have received a blow or other injury. The flat bones of the skull tend especially to undergo caries as a result of gummata



PLATE 37.—PUSTULAR SYPHILIDE.

*(Ricketts and Byles.)*



## SYPHILIS

infiltration, and some of the worst mutilations which result from syphilis and make it so dreaded are those due to caries of the bones entering into the formation of the nose and palate. Here, after a period during which the patient is afflicted by ozæna, the bridge of the nose may fall in, or a large perforation of the hard palate suddenly make its appearance. In other cases the process may spread from the underlying bone to the skin, and the most disfiguring ulceration of the face ensue. Gummata of the frontal and parietal bones may start in the inner or the outer table. In the latter case a swelling is followed by breaking down of the centre, and a circular or perhaps a horseshoe- or trefoil-shaped ulcer results. Here again the ulcer is rimmed with an ivory-hard ridge. Sooner or later the ulcer may reach the dura, but the general cavity of the cranium has usually by then become shut off from the affected area. When the gumma starts in the inner table, irritative and pressure symptoms may mark its first appearance. Gummata of the vertebræ are uncommon; according to their situation they may lead to retropharyngeal, lumbar, or iliac abscess. Rarely they may involve the spinal canal and cause symptoms of pressure on the cord.

Syphilitic **dactylitis** is very uncommon. Usually the proximal phalanx is affected, and a quiet, painless swelling results. Sometimes a sinus forms, and the bone becomes rarefied. In other cases the whole bone becomes permanently thickened, and in still others absorption of the process may result in permanent shortening of the phalanx.

Tertiary syphilis of the **testicle** occurs in two forms, which are often combined, the diffuse interstitial and the nodular. In the former the testicle slowly enlarges, becomes heavy and of indiarubber-like consistency, and the testicular sensation is lost. The nodular form, often engrafted on the diffuse, is characterized by the formation of gummata which project like bosses from the surface of the testis. The gummata may break down in the centre and eventually open through the skin, giving rise to a form of fungus testis.

The **mouth and throat** often suffer severely in tertiary syphilis. Gumma of the tonsil is followed by severe ulceration. The soft palate may be the seat of a condition analogous to the nodular cutaneous syphilide, being strewn with a number of pea-like nodules which often ulcerate and result in all grades of deformity; it becomes adherent to the posterior pharyngeal

wall, and in any case its pliability is considerably reduced. Perforations of the hard and soft palate may result from gummata commencing on the mouth side.

The **tongue** may be the seat of discrete gummata which eventually reach the surface and cause deeply punched-out ulcers. A much commoner manifestation of tertiary syphilis of the tongue is diffuse glossitis, which may be deep or superficial. The affected portions are swollen and the surface is smooth, hard, inelastic, and usually covered by a bluish-white pellicle (*leucoplakia*). On retrogression the tongue becomes cut up into numerous islands by fissures of varying depth. The tongue is tender, and the patient is intolerant of spices and hot food. (*See also TONGUE, SYPHILIS OF.*)

The insides of the cheeks, along a line from the angle of the mouth opposite the gap between the upper and lower teeth, are often affected with leucoplakia. The area forms a bluish-white ridge cut up herring-bone fashion which is quite characteristic.

**Diagnosis of tertiary lesions.**—An indolent swelling, or an ulcer which was preceded by a swelling, the lesion being obviously deeply embedded in the tissues or breaking down in the middle (which contains a characteristically gummy material) and sclerosing at the margin, with a circular, crescentic or evenly sinuous contour, and brownish red in colour, should always arouse a suspicion of syphilis. Too great stress should not be laid on denial of a primary sore, or of secondary lesions, which may long ago have been forgotten. The positive Wassermann reaction may mislead, since by no means all ulcers in an old syphilitic are themselves syphilitic. On the other hand, a negative Wassermann reaction is rather strong evidence against tertiary syphilis. *Epithelioma* is perhaps more likely to be confused with tertiary syphilis when the lesion is in the mouth, but epithelioma has a considerably harder margin and the edge is rolled, not clean cut. A positive Wassermann reaction may prove to be a trap, as epithelioma is often engrafted on an old syphilitic glossitis. Ulcers on the legs resulting from *varicose veins* or *impetigo* may arouse a suspicion of syphilis. They are usually much less regular in contour and associated with more inflammatory manifestations. Syphilitic orchitis is easily distinguished from other conditions by the evenly smooth hardness of the testicle, the weight, and the absence of pain or testicular feeling.

## SYPHILIS

**General constitutional symptoms of syphilis.**—Apart from the general symptoms indicating disease of special organs, certain constitutional disturbances call for some remark.

**Fever.**—Even in the incubation period observers have noted, in some cases, rigor followed by some degree of fever and pains in the limbs. In a certain proportion of cases, towards the end of the primary stage the patient's temperature may become irregular, the pyrexia being intermittent, continuous, or remittent, and accompanied by some constitutional disturbance. This pyrexia may not make its appearance until the rash is fully out. In some cases, just before the outbreak of the eruption and during the early part of the secondary stage, there is a feeling of aching in all the limbs, as if the patient had been taking too much exercise. This is often regarded as a natural accompaniment of the slight fever which is noted at the same time, but may possibly be due to meningeal involvement and irritation, which we now know is a fairly common event in the early secondary and later stages. Some support for this is found in a few cases which at this time suffer from definite lightning-pains. In the early secondary stage, also, headache may be severe, and, although possibly attributable to the accompanying pyrexia, may not improbably be due to meningeal involvement. In the tertiary stage fever has been noted in a number of cases, and its dependence on the tertiary lesion has been shown by its disappearance under antisypilitic treatment. The fever may simulate almost every type, and may be long-continued. It is by no means an invariable symptom of tertiary syphilis, but has been noted more particularly in connexion with syphilis of the liver. It may be mistaken for rheumatic fever in cases where there is arthritic pain due to periostitis in the vicinity of joints; for typhoid fever; and for tuberculosis. These examples may serve to show the type of fever and its long continuance in some cases, and point to the necessity of an investigation, with syphilis in view, in cases of obscure pyrexia.

**Anæmia.**—The organism of syphilis affects particularly the hæmopoietic organs, and it is not surprising that untreated syphilis is usually accompanied by some degree of anæmia. The anæmia is usually of the secondary type, with reduction of the red blood-corpuscles to an average between four millions and five millions per c.mm. The loss is more particularly displayed in the hæmoglobin content, which is usually reduced, so that the colour index may

be depressed to as low as 70 per cent. of the normal. The early stage is accompanied by a moderate leucocytosis which may reach a cell-count of 20,000 per c.mm. The increase of leucocytes is made up chiefly of lymphocytes, which is to be expected in view of the fact that the organism of syphilis stimulates a local exudate of lymphocytes and plasma-cells, of which the infiltration is largely composed. In pre-Wassermann days the blood-count constituted a not unimportant part of the investigation of a case of syphilis, though the features described above are by no means peculiar to this disease. In rare cases the anæmia may be much more severe, Müller having reported one in which the red blood-cells were reduced to 720,000 per c.mm., with 18 per cent. hæmoglobin, poikilocytosis and nucleated red cells; in fact, a blood-state resembling that found in pernicious anæmia. Justus, in 1895, introduced a new test based on the reduction of the hæmoglobin content which followed the first administration of mercury in a case of syphilis. He held that this phenomenon was observable, particularly from the date of onset of the secondary stage, and in all recurrences. Following the administration of 3 grm. of mercurial ointment by inunction or 1 gr. of mercury by intramuscular injection, examination of the hæmoglobin content on the two succeeding days showed a diminution which, in various workers' hands, has ranged from 8 to 35 per cent. It should be noted that the anæmia which is ordinarily found in syphilis is not attributable to the administration of mercury, since, after the preliminary fall in hæmoglobin, the remission of the disease under antisypilitic treatment is accompanied by an improvement in the blood picture. Opinions have differed on the value of Justus's test and it is now perhaps of only academic importance; but it may be of interest to observe that an explanation of the fall of hæmoglobin-content following the first administration of mercury may be found in the exacerbation of the activity of the spirochæta, which is known to occur after the first administration of any antisypilitic remedy. This increase in activity, reflected in an increase of symptoms and possibly in the conversion of a negative Wassermann reaction to positive, is known as the *Jarisch-Herxheimer reaction*.

### CONGENITAL SYPHILIS

As mentioned above, an infected mother can transmit syphilis to her offspring long after she has ceased to be sexually contagious. Syphilis

## SYPHILIS

transmitted to the foetus during the early months or years of the mother's disease is usually fatal, and an abortion or miscarriage results. If the disease has been in existence from one to three or four years, a child is born dead at full term. The next pregnancy may result in a living child which displays symptoms shortly after birth, and subsequent children usually show less and less marked signs, which tend to appear later, if at all, until eventually a child is born free from disease. The family history does not always pursue this regular course of gradual attenuation, since in some families an apparently healthy child may be preceded and succeeded by some miscarriages or stillborn children, but generally speaking the severity tends to diminish with the duration of the mother's disease. A possible explanation lies in the gradual decrease of the virus with which the infant is dosed and increase of the protective substances passed from the mother to the offspring.

**Symptomatology.**—A syphilitic infant born alive may show evidence of the disease in an eruption of bullæ or pustules resting on dark-red bases. These often break, and the eruption becomes crusted. The secretion contains the specific organism in large numbers. A commoner event is the maculo-roseolar eruption which comes on about three weeks after birth. It selects chiefly the napkin area, the neighbourhood of the nose and mouth, and the palms and soles, but the whole trunk and limbs may be affected. The macules are brownish red and tend to run together into large patches which are often annular, as in the recurrent form of acquired syphilis. In moist areas, and where the rash is exposed to friction, the surface secretes serum freely and becomes red and glazed, or crusted. Condylomata and moist papules occur as in the acquired form, and in the regions where skin and mucous membrane meet, as at the angles of the mouth and at the anus, ulcerative fissures or rhagades are apt to form. These radiate from the orifice and often leave scars which remain as stigmata of the disease until well on in life. Owing to the general interference with nutrition, syphilitic infants often become emaciated, with yellowish, papery skin and a wizened, old-man appearance. Simultaneously with the skin eruptions, the nails may become opaque and irregular. In others a severer form of onychia occurs, with infiltration of the nail and oozing of serum around the nail, which is loosened and shed. The hair of the scalp and eyebrows is often

shed extensively; on the other hand, syphilitic infants often show an abundant crop of hair, which has been called the "syphilitic mop," though it occurs in other diseases also. Mucous patches appear in the mouth and throat, and involvement of the nose and larynx is marked by the well-known "snuffles" and the hoarse, raucous cry of the syphilitic infant. Suppurative otitis media is often an early manifestation, and leads to hopeless deafness.

Tertiary lesions may make their appearance very early, or be postponed to the ages of 7, 14, 21, or even later. Thus diffuse interstitial orchitis may occur during the first six months. Skin gummata corresponding to the nodular cutaneous syphilide of the acquired disease may appear in the first year or two as small infiltrates which break in the centre to discharge their mucoid contents. These are the syphilitic furuncles of Barlow. Tertiary lesions of the mouth and throat may show themselves at any time after the first year, and result, as in the acquired form, in perforation of the palate and considerable deformity of the soft palate and faucial pillars, from ulceration and subsequent scarring. Destruction of the nasal support results in saddle-nose, and from the age of about 11 years there is a liability to sudden deafness from guminatous changes of the internal ear. The affection usually starts in one ear but quickly follows in the other, and the child becomes permanently deaf. Choroiditis and iritis often appear as early as the age of five months, and, unless properly treated, lead to blindness or considerable impairment of sight. Interstitial keratitis may begin at the age of 6 or be postponed until the patient is between 20 and 30. Usually it starts in one eye and the other follows suit in a few months, often in spite of treatment. The cornea of the affected eye gradually becomes opaque from the margin inwards, and this is followed by vascularization, which results in the formation of a pink patch ("salmon patch"); then the cornea may gradually weaken and bulge before the intraocular pressure, or it may ulcerate. Gradually, after many months the cornea may clear more or less completely, but the patient is often left with permanently damaged sight owing to concomitant iritis and choroiditis. The permanent teeth may show signs of congenital syphilis in the form of notching of certain teeth. The two upper central incisors are much the most usually affected, but all the incisors may be narrowed at their cutting bases, which are also concave (*Hutchinson's teeth*).

## SYPHILIS

These characteristics are usually lost after the age of 20. The first molars are often dome-shaped, the cusps having fallen together from failure of the central pillar to mature (*Moon's teeth*).

The long bones may be affected in various ways. During the first few months syphilitic epiphysitis may cause signs simulating paralysis (*syphilitic pseudo-paralysis*) owing to the rapid loss of movement which occurs. Movement of the limb causes severe pain, which explains the apparent paralysis. The epiphysis is swollen, and occasionally definite separation occurs.

Dactylitis occurring in the second year causes fusiform swelling of the proximal phalanges of two or more fingers or, more rarely, toes. At a later period in childhood, between the ages of 8 and 14, chronic periostitis of long bones is apt to cause characteristic deformities, which are best shown in the tibiae. These are lengthened, increased in breadth from before backward, and the curving forward of the crest produces the appearance of a sabre-scarbhard.

Arthritis may be associated with the epiphysitis mentioned above, and the joint may suppurate. Between 5 and 18 years of age chronic synovitis affecting chiefly the knees, with swelling of joints but with slight pain or interference with movement, may be puzzling to diagnose unless syphilis is remembered. Chronic osteo-arthritis simulating the disease in adults may also occur.

Visceral forms of congenital syphilis are considered in other articles.

The nervous forms correspond generally with those of acquired syphilis. The use of the Wassermann test has shown that many nervous affections of children and adolescents have a syphilitic origin, which was formerly unsuspected on account of the absence of stigmata. This applies particularly to mental deficiency and epilepsy, in which some observers, examining the blood-serums of groups of children, have found positive reactions in as high as 40 per cent. or more. It is noteworthy that obvious stigmata were found in only about a fifth of these cases. Juvenile tabes and general paresis, though very uncommon, have to be remembered as possible results of congenital syphilis. The admissions to asylums for general paresis of persons under 25 in the years 1908-12 were 81 males out of a total of 5,352, and 44 females out of a total of 1,028.

**Diagnosis.**—This is not difficult if the observer remembers the existence of such a disease and has a Wassermann test carried out. Syphilitic pemphigus occurs on the palms and is earlier than ordinary *pemphigus neonatorum*, and *Sp. pallidum* can be found in the secretion. Ordinary *erythemata* are brighter red and associated with friction and moisture such as from wet napkins; the maculo-roseolar syphilide is not necessarily accounted for by friction or by moisture, occurring anywhere, and is associated with such signs as "snuffles" and chronic laryngitis.

Syphilitic epiphysitis is distinguished from *true paralysis* by the pain on movement and contraction of the muscles when the skin is irritated. It occurs earlier than *scurvy* or *rickets*, appearing about the third week, and is associated with the skin and mucous-membrane lesions of syphilis.

### PROPHYLAXIS OF SYPHILIS

The virus of syphilis is undoubtedly very delicate, and the prompt application of any antiseptic should suffice to prevent infection. In a classical experiment on man, Metchnikoff prevented the development of syphilis, after inoculation with virulent syphilitic virus, by rubbing the following ointment into the scarified area an hour after the inoculation :—

Calomel . . . . .	33
Lanolin . . . . .	67
Vaselin . . . . .	10

A monkey rubbed with the same ointment twenty hours after inoculation developed syphilis. In a subsequent experiment syphilis was prevented in a monkey by rubbing the inoculated area with the ointment eighteen hours after the inoculation. It would not be safe, however, to rely on this later experiment in the case of man, as monkeys are much less susceptible to syphilis than men. Probably the most effective method would be to steep the part for some minutes in 1-in-2,000 perchloride of mercury and then to rub in the above ointment.

The prevention of accidental infection of fingers, lips, and other extragenital parts resolves itself into measures to prevent the direct or almost direct contamination of abrasions with the virus from syphilitic persons who are in the earlier stages of syphilis. As the virus, apparently, does not long remain virulent outside the body, it is probable that articles contaminated with secretion from

## SYPHILIS

syphilitic sores must soon become safe for use without further precaution; otherwise accidental syphilis would be rife, considering the numbers of syphilitic persons with mouth secretions teeming with *Sp. pallidum* who frequent public restaurants and reside in the midst of their families without taking any special precautions to prevent the disease being passed on to others. Such persons should, however, be warned of the risk arising from their sharing of table utensils, crockery, and house linen with others. They should not kiss others nor talk directly into people's faces, and articles which they have used should be dipped into very hot water. A further precaution is to keep open lesions smeared with an antiseptic ointment such as the Metchnikoff cream mentioned above.

The prevention of transmission to offspring is primarily a matter of preventing infection of mothers. A man who has contracted syphilis should be advised to wait for at least four years before marrying; by that time the risk of transmission by sexual intercourse has almost always gone, whatever the treatment. The period can be shortened by adequate treatment, but it must be treatment which is calculated to give 100 per cent. of cures, and that cannot be claimed for the treatment usually administered at the present time. A woman who has contracted syphilis is liable to convey the disease to her offspring for a considerably longer period than that in which she remains a danger through sexual intercourse, but efficient treatment before or during pregnancy will prevent the disease being transmitted, or, if it has already been transmitted, will go far towards curing the infant before it is born. Naturally, the earlier the treatment is commenced the more likely the success.

### TREATMENT OF SYPHILIS

The remedies most commonly employed for the treatment of syphilis are preparations of arsenic, mercury, and iodine. The first two are generally believed to have a destructive action on the parasites, the last is believed to act on the tissues, stimulating them to clear away granulomatous tissue.

**"606" and its modifications.**—Arsenic has been used for the treatment of syphilis for a very long time, but played a subsidiary rôle until 1909, when Ehrlich produced "606" or dioxydiamidoarsenobenzol dihydrochloride, which is now sold as salvarsan, arsenobenzol (Billon), arsenobillon, kharsivan, diarsenol,

and arsphenamine, according to the country of origin and the manufacturing firm. It is a yellow powder which is readily soluble in water, in which it forms an acid solution. Addition of an alkali to this solution causes a precipitate which redissolves on addition of more alkali. Being liable to change on exposure to air, it is sold in sealed ampoules containing weighed amounts of the compound. It was at first administered intramuscularly, but caused so much pain that this method was abandoned and the intravenous route employed. For intravenous injection it is necessary to convert the acid compound to the alkaline, since the acid solution causes severe vaso-motor symptoms, probably on account of the compound being precipitated too rapidly by the alkaline blood. The following is a good method of preparing the remedy for intravenous injection:—

1. Inspect the ampoule to see that it is intact.
2. Dissolve the contents in germ-free, metal-free distilled water at the rate of 0.1 gm. in 10 c.c. of water contained in a tall, stoppered glass cylinder, which is shaken until solution is complete.
3. Add 0.8 c.c. of 4-per-cent. sodium-hydrate solution for every 0.1 gm. of the remedy. The solution becomes at first generally turbid and then perfectly clear.
4. Add 0.5-per-cent. sodium-chloride solution to make the strength not greater than 0.1 gm. in 25 c.c.
5. Test with litmus as a check on the correctness of the above technique.
6. Strain through sterile muslin or many layers of gauze.
7. Administer intravenously without delay.

The dose for normal adults varies from 0.3 to 0.6 gm., so that not less than 75 c.c. fluid has to be administered, and a special apparatus is necessary. This may be a syringe, by which the solution is alternately drawn from a receptacle and forced into the vein; or a bottle, from which the solution is forced along a flexible tube to the needle by pumping air into the bottle above the solution; or the solution is allowed to flow by gravity from a funnel held above the patient. The choice of apparatus depends on the taste of the operator; my preference is for the gravity method. It is advisable, after inserting the needle, to allow a little saline to flow first, as a precaution against the injection of "606" into the tissues should the needle have happened not to enter the vein. Some saline should be run in afterwards to clear the needle of



## SYPHILIS

the "606" solution. For this purpose it is convenient to contain the "606" in one funnel and the saline in another, both funnels being slung side by side and connected, about a foot below, by a glass V-piece. After the needle has been inserted, the clip controlling the saline is opened, and as soon as it is seen that the flow is satisfactory the "606" solution is turned on while the saline is shut off. The saline is turned on again, as soon as sufficient "606" has flowed down the tubing, to sweep that remaining in the tube into the vein. The whole procedure is very simple, but requires rather an elaborate set of apparatus and uses up so much distilled water that a simpler technique is likely to appeal to the practitioner. This is not possible with the original preparation, which must be given well diluted, and Ehrlich experimented to discover a compound having the same therapeutic properties but more convenient to administer. The result was two preparations, one of which was produced shortly after "606" and is now almost universally used; the other, produced not long before the War, has not been employed very extensively in this country. The second of these preparations, "**salvarsan-natrium**" or "**sodium salvarsan**," is "606" already converted to the sodium compound. It is a yellow powder which requires only to be dissolved in water and can be given much more concentrated than the original preparation. It is about two-thirds the strength of salvarsan and, in equivalent doses, is a more easily tolerated preparation than the original.

The first improvement of salvarsan produced by Ehrlich was "**914**," or dioxidyamidoarsenobenzol monomethylene sulphonylate of soda, which is now sold as neosalvarsan, novarsenobenzol (Billon), novarsenobillon, neokharsivan, neodarsenol, and neo-arsphenamine. It is a yellow powder (containing 18 to 22 per cent. of arsenic) which is much more liable than salvarsan to become toxic on exposure to air, so that it must be administered very quickly after the ampoule has been opened and its contents dissolved. The powder is soluble in its own weight of water (samples which dissolve with difficulty should not be used), in which it forms a neutral solution. The strength of this compound is two-thirds that of "606," a dose of 0.6 corresponding to 0.4 of the original preparation. The most usual method of administration is the intravenous. The dose, from 0.45 to 0.9 for adults, is dissolved in 2-5 c.c. of distilled water and drawn into a

syringe through a cotton-wool filter or after being drained through muslin. The syringe is armed with a fairly fine needle, and the point of the latter inserted into the vein, which has been made prominent by fastening an elastic band round the upper arm and making the patient grasp a roller bandage. A pull on the piston causes blood to flow back into the syringe when the needle is properly within the vein. The rubber band is released and the hand opened, and the piston is then pressed steadily home. "914" can also be given intramuscularly or into the deep subcutaneous tissues, and its therapeutic effect when administered in this form is undoubtedly greater than when injected intravenously. The injection is apt to cause considerable pain, which may be immediate or may come on two or three days later and last for about a week. Many methods of preparing the solution have been devised to obviate the disadvantage, but none of them has proved entirely successful, and many workers consider that simple solution of the dose in about 10 minims of distilled water results in as little discomfort as when such anæsthetics as stovaine, creocamph, and camphophenique are added. Recently a modification of "914" has been prepared by Pluchon which can be injected subcutaneously with practically no discomfort to the patient. It is known as "**sulfarsenol**."<sup>1</sup> It is a yellow powder which is readily soluble in distilled water and can be injected in a concentration of 0.3 grm. per c.c. of water. If injected intramuscularly it is apt to cause some pain and aching in the muscles, though this appears to be less than that which often follows an intramuscular injection of mercury. The best method of administration is into the deep subcutaneous tissues. In the upper and outer quadrant of the gluteal region the skin and fat are pulled away from the underlying fascia, by grasping them with the thumb and fingers of the left hand, and a 2-in. record needle is entered obliquely at the base of the pyramid thus produced. The needle is made to under-run the fat so that its point may scrape on the fascia overlying the gluteal muscles. The charged syringe is fitted to the needle and the injection given fairly slowly. The site is then massaged with a pad of lint.

Another preparation, introduced by Monneyrat, is **galyol**, or tetraoxydiphosphamino-diarsenobenzol. It is a greenish-grey powder

<sup>1</sup> Sulfarsenol is sold in London by Messrs. Wilcox, Jozcau & Co., chemists, Haymarket.

## SYPHILIS

which is soluble in alkali, and is sold in sealed ampoules already mixed with sodium carbonate, so that it is ready for intravenous administration when the contents of the ampoule have been dissolved in about 5 c.c. of distilled water. Galyl is also sold dissolved in glucose solution for intramuscular injection.

Two preparations have been introduced by Kolle, the successor of Ehrlich. One, called "sulfoxylate" or "1495," is a stable arsenobenzol preparation put up in ampoules already dissolved and ready for injection. The other preparation, known as "silbersalvarsan," is a combination of salvarsan and silver and is closely similar in its physical properties to "disodo-luargol," a combination of salvarsan, silver, and antimony, which was introduced by Danysz in 1916. A preparation made by the same firm and known as "arseno-argenticum" appears to be identical with silbersalvarsan. It is an emery-coloured powder which forms a dark-brown solution in water and can be given intravenously in solutions as concentrated as 0.3 gm. in 10 c.c. A good method of giving these silver preparations of salvarsan is to dissolve the dose in 5 c.c. of distilled water, take up through a cotton-wool filter into a 10 c.c. syringe and, having punctured the vein, to draw into the syringe 5 c.c. of blood before pressing the piston home. This ensures the correct position of the needle; the blood cannot be seen to enter the syringe barrel owing to the opacity of the solution, but the piston could not be pulled back so far if the needle were not properly within the vein. The injection should be given much more slowly than neosalvarsan, otherwise the patient may suffer from vasomotor symptoms as noted below. When the vein is a very difficult one it may be better to inject in more dilute form with a funnel and rubber tubing, as used for the administration of salvarsan or its substitutes. The dilute method may also obviate thrombosis, which occurs sometimes after using more concentrated solutions.

**Therapeutic properties of arsenobenzol preparations.**—All the arsenobenzol preparations mentioned above have a very rapid effect in causing disappearance of *Sp. pallidum* and of symptoms. In this respect there is little to choose between them, but Kolle's sulfoxylate is rather slower than the others. All of them act much more rapidly than mercury, and there is no doubt that the use of these preparations has considerably improved the treatment of

syphilis, though the first hope of Ehrlich that he had discovered a remedy which would cure with one dose has not been fulfilled. They have, moreover, very considerably improved the prognosis. Whereas before, under purely mercurial treatment, it was common to see a patient develop new symptoms whilst actually taking the remedy, it is now rare for anyone to relapse whilst undergoing a course of arsenobenzol treatment. The rapidity of effect can best be appreciated in cases where it is necessary to stop the syphilitic process quickly, before it has done irreparable damage to delicate structures such as the iris; in such cases these remedies are incontestably superior to mercury. The greater certainty of cure is shown by the fact that unequivocal second attacks of syphilis, formerly a great rarity, are now of comparatively common occurrence, even in the experience of those who understand the difference between recurrent and new chancres. Dose for dose, luargol, arseno-argenticum, and Kolle's silver salvarsan appear to be about twice as powerful in immediate therapeutic effect as "606" and "914," and can consequently be given in much smaller doses. Sulfoxylate is slower in action. In permanence of effect these compounds differ somewhat. Of the three better-known preparations, "606," "914" (and their respective substitutes), and galyl, experience indicates that, in equivalent courses administered intravenously, the permanence of effect is in the order given, i.e. after a given course the smallest percentage of relapses will follow the "606" and the largest the galyl. When the results of an intramuscular or deep subcutaneous course of neosalvarsan are compared with these there is little doubt that the advantage rests with neosalvarsan, and it is reasonable to expect that the same will be found of the closely similar preparation, sulfarsenol. Sufficient is not yet known of luargol, arseno-argenticum, silver salvarsan, and sulfoxylate to indicate their position in regard to permanence of effect, but considerable evidence has been produced to show that the silver arsenobenzol preparations are particularly valuable in severe ulcerative conditions of syphilis and in syphilis of the nervous system.

All these remedies have a certain toxic effect on the tissues, which is manifested in a small proportion of cases by symptoms described below. On the whole, "606" appears to give rise to immediate side-effects in a larger proportion of cases than the others. As a practical

## SYPHILIS

result, patients have to be kept under observation for some hours after salvarsan injections—a precaution which is not, as a rule, necessary after injections of the other remedies. The choice of preparation depends on circumstances. Other things being equal, the worker would probably choose “606” for routine intravenous and sulfarsenol or neosalvarsan for subcutaneous treatment because of their superior therapeutic effect, though it is possible that these may be ousted later by the silver combinations when we have had sufficient time to test these newer remedies. But salvarsan has disadvantages in the complexity of its preparation, in the length of time consumed over the injection, and in the fact that the patient has to be detained for some hours; while “914,” on the other hand, is simply prepared, and the patient can be sent away at once. The first is therefore a preparation for intravenous treatment of in-patients at an institution where assistance and appliances are ample; the second is for the consulting room and the out-patient department. For subcutaneous treatment sulfarsenol is at present the remedy of choice.

**Toxic effects of arsenobenzol remedies.**—These compounds all tend to damage capillary endothelium, and most of the toxic effects can be referred to this action. In patients who have died as a result of over-doses, or of idiosyncrasy, there have been found blockage of cerebral capillaries with small hæmorrhages around these; hæmorrhagic nephritis; hæmorrhage into lung capillaries; submucous petechiæ and ecchymoses in the stomach and bowel; and, in a comparatively few cases, degeneration of liver cells amounting to a condition like that found in acute yellow atrophy.

Clinically, toxic effects are manifested by one or more of the symptoms now to be enumerated. Although the list is a comparatively long one, most of the symptoms are so mild, infrequent, or preventable as not to preclude the routine use of these remedies. In roughly chronological order they are as follows:—

**1. During or immediately after the injection:—**

- (1) Vaso-motor disturbances, also known as anaphylactoid symptoms or minor nitritoid crises. (2) Urticaria. (3) Syncope. (4) Pain in the gums and teeth. (5) Peculiar taste in the mouth.

**2. Following the injection usually by a few hours, and occurring generally on the same day:—**

- (6) Rigor, rise of temperature, and headache. (7) Vomiting, diarrhoea, pain in the back and cramp in the legs. (8) Herpes (labialis or zoster).

**3. At various times from a day or two to a month or longer after a single injection or a course of injections:—**

- (9) Albuminuria. (10) Stomatitis. (11) Chronic headache; lassitude; loss of appetite, weight, and sleep. (12) Erythema and dermatitis. (13) Jaundice. (14) Severe cerebral symptoms.

1. The *vaso-motor symptoms* simulate those of anaphylaxis so closely as to have been attributed to that condition. The face becomes flushed, and the tongue and lips may swell; there may be respiratory distress, and the patient may become unconscious. Often a severe attack is followed by more or less generalized *urticaria*. As a rule the symptoms last for about half an hour, but in rare cases recovery is not complete for a number of hours. There is no doubt that they are due to the remedy entering the circulation in an imperfect state of solution or being precipitated too rapidly in the blood-stream. Samples of “914” which dissolve with difficulty cause these symptoms when given in concentrated form, though the same samples are innocuous when given dilute. Imperfect alkalization of “606” solutions, their very rapid administration, or (what amounts to the same thing) giving the injections in too concentrated a form, almost always results in vaso-motor symptoms. All produce greater density of the precipitate in the blood-stream. Some patients react more readily than others, and this is possibly related to the effect of their blood upon the physical state of the preparation.

The prevention of vaso-motor symptoms depends on attention to the above considerations. Solutions of “606” should be well alkalized and given dilute; solutions of “914” should not be given concentrated if the preparation does not dissolve perfectly in practically its own weight of water; all solutions should be filtered before use. The treatment usually employed is to inject 10-15 min. of adrenalin chloride (1 in 4,000) hypodermically, but atropine  $\frac{1}{10}$  gr. appears also to be effective.

*Syncope* is usually mental, unless it precedes vomiting.

*Pain in the gums and teeth* is probably vaso-motor.

As to the *peculiar taste in the mouth*, of which

## SYPHILIS

some patients complain during the injection, this has been interpreted by some authors as a sign of intolerance of the remedy. I have noted the presence or absence of this symptom in almost every patient to whom I have given an arsenobenzol preparation, and cannot agree that it indicates intolerance. It is a very common symptom when "914" is given concentrated, and far more frequent than when "606" is given dilute, yet one would say that toxic effects are commoner after the latter. Patients of mine who have received exceptionally large total amounts of "606" without ill effect have tasted it every time, whilst amongst those who have never tasted the more concentrated "914" there have been as many who developed signs of intolerance as among those who did experience the peculiar taste.

2. *Rigor, rise of temperature, and headache* are very rarely severe, but are commoner after first than subsequent injections. *Diarrhœa* and *vomiting* are not frequent unless there has been an error in technique, or the patient has been indiscreet in his dietary. Quite often they are followed by *herpes*. Usually these symptoms have all disappeared by the next day. They are prevented to some extent by taking care that the patient has fasted for some hours before the injection.

3. *Albuminuria* very rarely causes any anxiety. *Stomatitis* is not often attributed to arsenobenzol remedies, but these undoubtedly seem to increase the tendency to this complication which is manifested by patients on mercurial treatment.

*Chronic headache, lassitude, etc.*, are symptoms of intolerance displayed by a few patients, and indicate the necessity of a rest from treatment.

Various *skin affections* may occur besides the urticaria and herpes mentioned above, and may have very serious consequences. The mildest is some slight itching which quickly passes off. Some patients show a transient and limited erythema which quickly disappears, but in a small minority a punctiform erythema quickly spreads over the body, is accompanied by most intense itching, and often then passes on to a condition like pityriasis rubra. There is extensive exfoliation of the epidermis and localized weeping eczema. The constitutional symptoms are severe, with fever, insomnia and loss of appetite, and there may also be severe diarrhœa, perhaps with jaundice, indicating intestinal catarrh. The incidence of dermatitis depends largely on the intensity of the

treatment. Generally speaking, a male adult of average build will easily tolerate 2.6 gm. of salvarsan in doses of 0.3-0.5 spread out over a period of 57 days, but if this period is shortened the percentage of dermatitis increases noticeably. This was a consideration in mapping out the course of treatment outlined later (p. 320). There is no doubt, also, that a careful look-out for signs of skin irritation will often supply timely warning of the idiosyncrasy and, by preventing the administration of more arsenobenzol will save the patient from a severe attack. The treatment of exfoliative dermatitis following arsenobenzol injections is often troublesome on account of the generalized exfoliation and the local pustulation and eczema. Generally, the patient should be in bed and well protected. An intramuscular injection of intramine 2.5 c.c. will often abort a commencing erythema, as also will bleeding to a pint. Phlebotomy is useful, too, in relieving the headache and general irritation. Internally, quinine 5 gr. every four hours, and ichthyol 5 gr. in capsule night and morning, are useful, and the bowels should be regulated with paraffin. The diet should be simple, and should not include eggs and meat. Locally, calamine lotion and starch poultices are soothing. An occasional bran bath is valuable, but careful precautions should be taken against chill, as these patients are very prone to pneumonia. For weeping surfaces I prefer 10-per-cent. ichthyol ointment.

*Jaundice* following injections of arsenobenzol preparations has been the subject of considerable discussion, and views as to its causation are by no means unanimous. In a series of 39,377 patients treated with "606" or "914," I found that 270 had been reported as suffering from this complication either during or some time after the course of treatment. The patients suffered no more severe symptoms than are met with in the average case of obstructive jaundice, though in some instances the symptoms persisted for several months. In 24 cases the symptoms were definitely of a different type, with severe epigastric and hepatic pain, restlessness and delirium, followed by death. Broadly, the changes found were extensive degeneration of liver-cells with round-celled infiltration of the supporting connective tissue; multiple subserous hæmorrhages; and frequently hæmorrhages into lung alveoli. No particular batch of the preparations could be incriminated as responsible for these fatalities, which occurred in three hospitals, nor could

## SYPHILIS

the technique be called in question. They happened about the same season, and it is significant that, while the hospitals concerned were dealing constantly with about 1,000 cases of syphilis, seven other hospitals dealing with more than twice this number experienced no instance of acute toxic jaundice. As far as could be ascertained by personal inspection, the technique was practically the same in all the hospitals. As to dosage, at Rochester Row, where on an average more of the remedy was administered per patient than at any other hospital, no case of the kind occurred, but it seems significant that, in the two hospitals which suffered worst, the treatment was concentrated into a shorter period for the same total dosage than in the others. It has been suggested that the jaundice is primarily syphilitic in origin and that the remedy completes the destruction of the damaged cells, either by direct action or by releasing toxins from the spirochaetes. This probably does account for some cases. But the treatment was common, and syphilis was common to all the hospitals, yet these cases occurred in only three of the eleven hospitals which contributed the above collection of 39,377 cases. After allowing for the factor of concentration of the course of treatment, which has been mentioned above, it is difficult to escape the impression that another factor was concerned in the causation—some agent which damages liver-cells and makes them an easy prey to the toxic effect of the arsenobenzol remedy. It is not unlikely that the agent of campaign jaundice was concerned in many of these cases, and this would explain its epidemic character. It is obvious, however, that the arsenobenzol compound played a considerable part as precipitating agent. It should be mentioned, by the way, that the incidence of toxic jaundice ought to be put at a much lower figure than is represented by 24 out of 39,377, since these were collected only over a certain period. Up to the early part of 1919 I had gathered reports of only 30 fatal cases of jaundice from military hospitals treating syphilis in the United Kingdom during the War. These hospitals must have dealt with more than 60,000 cases of syphilis.

The treatment of both types of jaundice mentioned above consists in milky diet without meat or eggs, and large doses of bismuth subcarbonate and sodium bicarbonate, with regulation of the bowels by paraffin.

Severe *cerebral symptoms*, headache followed by mental confusion, epileptiform convulsions

and coma, ending in death in a large proportion of cases, are now very rare, and I was able to discover only eight cases among soldiers treated in Britain before and during the War. Such cases were much more frequent in the early days of salvarsan, when it was common for two injections of 0.6 grm. to be given with an interval of forty-eight hours, and there can be no doubt that the comparative infrequency of cerebral side-effects among cases treated in military hospitals has been due to the moderate initial dosage employed and the spacing out of the injections. One patient, whom I saw in the comatose condition, was restored within half an hour by phlebotomy to 20 ounces, the removal of 15 c.c. of cerebro-spinal fluid, and the injection of 1 c.c. of adrenalin chloride 1 in 1,000. This treatment should be applied at once.

**Jarisch-Herxheimer reaction.**—The immediate effect of these as of other antisyphilitic remedies is to increase the intensity of the syphilitic process. Generally this is of no great moment, as when a rash becomes more intense, but it may be important when an artery supplying some vital organ is already partly blocked. Such cases, however, are very rare.

**Neuro-recurrences.**—Though not strictly a direct effect of arsenobenzol treatment, paralysis of various cranial nerves, especially the seventh and eighth, became more common shortly after its introduction. These phenomena have been proved to be syphilitic recurrences, and their greater frequency at one time was due to the fact that many patients were treated with one or two doses only, in the belief that that was sufficient to cure. It seems probable that the arsenobenzol remedies prevent development of the spirochaetes in the skin and mucous membranes more easily than in the central nervous system, and that the absence of any general skin and mucous-membrane reaction results in failure of development of immune substances which would keep the spirochaetes in the central nervous system in check. If this is so, the physician clearly must administer an amount of treatment which will ensure the destruction of spirochaetes in the central nervous system as well as the other tissues. If he stops short of this he may give an opportunity to the spirochaetes in the brain and spinal cord to set up greater trouble than would otherwise be possible.

**Precautions recommended in treating patients with arsenobenzol compounds.**—In

## SYPHILIS

the case of sufferers from advanced Addison's disease, bleeders, and those on the point of death from severe visceral disease, these remedies are contraindicated.

In visceral disease of a less severe type, in alcoholism, and when the patient is prone to such skin affections as eczema and severe seborrhoea, it is advisable to begin with a dose of 0.1-0.2 grm. of "606" or 0.15-0.3 grm. of "914," and to increase the intervals in the outline of treatment given in the next paragraph by from 20 to 100 per cent. The same applies to patients suffering from syphilis of the brain, cord, or viscera when there is reason to fear an exacerbation of the process.

In the case of adults who, except for syphilis, are healthy, it is well to remember that the most healthy-looking may be intolerant of the remedy, and to begin with a moderate dose—0.3 grm. of "606" or 0.45 grm. of "914." On the same principle the intervals between doses should be spaced in a way which has proved to result in a very low proportion of toxic side-effects. I have tried various courses, from 2.4 grm. of salvarsan in 28 days, gradually increasing the intervals until 2.6 grm. was being given in 57 days, and have found in the case of men that when 2.6 grm. is compressed into less than a 57-day course the incidence of such toxic side-effects as dermatitis increases noticeably. The patient should be watched carefully throughout the course for the signs of intolerance mentioned above. This precaution may not prevent severe side-effects absolutely, but such as do develop will usually be much milder than when no notice is taken, for example, of an erythema and the treatment is continued to the end of the course.

**Mercurial preparations.**—Mercury was formerly the sheet anchor in the treatment of syphilis. Since the advent of the arsenobenzol preparations it has largely been relegated to the second place, and even discarded altogether by some, who believe that a cure can be effected more rapidly and certainly by the newer preparations alone. My own experience is that more relapses occur after the administration of a given number of injections of arsenobenzol alone than when as many injections of an insoluble preparation of mercury are given also, at the same time. Mercury acts slowly, and the therapeutic dose is very close to the maximum which the patient can tolerate, so that its limitations are particularly apt to be felt when

it is necessary to bring the disease rapidly under control, as in iritis and in cases where the patient is rather less tolerant than normal, so that sufficient of the remedy cannot be administered to control the disease. It has the advantage over the arsenobenzol preparations that it can be kept in the circulation almost continuously for years. That one dose of arsenobenzol, even a large one, does not cure syphilis is now well known. It seems probable that the spironemes most deeply placed in infiltrates are not reached, and that before the last spironeme can be destroyed the infiltrate must be removed and the area revascularized. Such a process takes time, and whilst it is occurring it is necessary that spironemes should be kept in check as they become exposed; otherwise they would be set free to start the process of syphilization anew. This conception indicates the rôle of mercury in the modern treatment of syphilis. If, however, the view is held that the disease is not cured by one dose of arsenobenzol because the spironemes are in a resistant form, it is equally reasonable to hold that, as they resume the form which is susceptible to attack by antisypilitic remedies, one of these ought to be present in the tissues to keep them in check; and it is mercury that can most conveniently be maintained in the tissues for this purpose.

**Methods of administration.**—The oral method is much favoured, but is apt to cause gastrointestinal disturbance and is exposed to the risk of failure through the patient's forgetfulness. It is greatly employed now for continuation treatment after the patient has undergone sufficient arsenobenzol and mercurial treatment to render his Wassermann reaction negative. The favourite preparations are: (1) Hydrargyrum cum creta, with or without pulv. ipecacuanhæ composita, 1-2 gr. of each in pill or tabloid form; (2) hydrargyrum iodidum viride,  $\frac{1}{4}$ - $\frac{1}{2}$  gr. in pill form; (3) pil. hydrargyrum, 1-3 gr.; (4) liq. hydrargyrum perchloridi,  $\frac{1}{2}$ -1 dr., often prescribed in a mixture with potassium or sodium iodide. The fourth preparation is quicker in effect than the other three in corresponding doses, but is obviously less convenient for the patient, since it involves the carrying about of a liquid mixture.

A good method of planning continuation treatment, taking hydrargyrum cum creta as the basis, is as follows, the courses of arsenobenzol and mercurial treatment outlined later (p. 322) having been completed:—

## SYPHILIS

- (1) Pil. hydrarg. c. cret. 2 gr. thrice daily for one month, followed by three days without pills.
- (2) Pil. hydrarg. c. cret. 1 gr. four times a day for one month, followed by one week without pills.
- (3) Pil. hydrarg. c. cret. 1 gr. thrice daily for one month, followed by one month without pills.
- (4) Pil. hydrarg. c. cret. 1 gr. thrice daily for three months, followed by one month without pills.
- (5) Pil. hydrarg. c. cret. 1 gr. thrice daily for three months, followed by one month without pills.

During the intervals of one month it is advisable to give iodides in the doses specified (p. 321).

**Inunction.**—This is a most valuable method of administering mercury but must be carried out by a skilled rubber. Inunctions have the inconvenience of soiling the skin and clothes. On successive days 5–10 grm. of 33½-per-cent. mercurial ointment is rubbed for twenty minutes into thighs, calves, arms, and back, the number of rubbings varying from 60 to 200 in a course. The length of a course depends on the patient's tolerance, which is judged by the state of the gums, the weight, and the general well-being.

**Intravenous injections** are rapid in effect but very apt to give rise to toxic symptoms; they appear to me to have the further disadvantage that the effect is not sustained. The usual preparations employed are the cyanide and the perchloride, in doses of 1 c.c. of the 1-per-cent. solution, daily or on alternate days.

For **intramuscular injections** both soluble and insoluble preparations are employed. Among the *soluble* preparations are the biniodide (1-per-cent. solution) in doses of 1 c.c., the bibromide, the benzoate, and the perchloride, the perchloride being made up as follows:—

Sod. chlor. . . . .	gr. 4
Aq. destill. . . . .	℥400

Dissolve, filter, and add hydrarg. perchlor. gr. viii.  
Dose, 5–10 min.

The soluble preparations are more rapid in effect, but this is not sustained. They have the further disadvantage of having to be given daily or on alternate days.

The *insoluble* preparations most commonly employed are mercury in fine subdivision, calomel, and mercury salicylate. The two former are suspended in a fatty base, such as palmitin and creocamph, or olive oil, in strengths of 10, 20, or 40 per cent., according

to preference. Mercury salicylate is usually put up in a 10-per-cent. suspension in liquid paraffin. Suspensions of these remedies ready for use are sold by most chemists. The doses usually employed are mercury 1–1½ gr., calomel ½–¾ gr., salicylate 1½–2 gr. Calomel causes more pain than the other two, and my own preference is for mercury in fine subdivision. The advantages of the insoluble preparations are that, on account of the slow absorption, enough mercury can be given in one injection to last a week, and that the effect is sustained, so that the proper rôle of mercury in the modern treatment of syphilis is fulfilled.

The technique of intramuscular injections is simple. The site usually chosen is the gluteal. A good point is just external to the mid-point of a line drawn from the meeting point of the buttocks to the top of the great trochanter. A needle 2 in. long is introduced, almost to its full length, perpendicularly to the skin surface. The base is examined to see that no blood is oozing from it, the syringe is applied, the piston pulled upon to see that the needle point is still not within a vein, and the piston is then pressed home. The needle having been withdrawn, the site is well massaged with a ball of cotton-wool. Before an insoluble preparation is drawn into the syringe, care should always be taken to see that the suspension has been well mixed, either by stirring with a glass rod or by energetic shaking of the bottle.

**Toxic effects of mercury.**—These are stomatitis, nephritis, colitis, general malaise, and dermatitis.

**Stomatitis** can usually be prevented with care. The patient's teeth should be set in order before starting the course, and he should brush his teeth night and morning. Potassium chlorate is useful and may be incorporated in the dentifrice as follows:—

Ry	Cret. præcip. ʒi.
	Mag. carb. pond. ʒiii.
	Sap. pulv. dur. ʒiii.
	Thymol gr. ii.
	Potass. chlorat. gr. xvi.
	Ol. menth. pip. ℥xv.

If in spite of these precautions the gums become sore, the mercury must be stopped and more energetic treatment applied to the mouth. Lozenges of potassium chlorate to suck, and swabbing with peroxide of hydrogen, followed by the application of collosol argentum, or of a mixture of liq. arsenicalis 1, vinum ipecacuanha 1, and spiritus vini rectificatus 2, usually suffice

## SYPHILIS

to restore the gums to a healthy condition. Between times a mouth-wash of alum, 5 gr. to the ounce, or of liq. plumbi subacetatis dilutus is valuable. These measures can usefully be supplemented by the administration internally of sulphur water and of potassium iodide.

*Nephritis* rarely results from the moderate doses of mercury now employed, but the effect of mercury as an irritant of the kidneys should be remembered in cases where these organs are already diseased. Arsenobenzol preparations also tend to cause nephritis, so that, in certain cases, it may be advisable to withhold one preparation during the period when the other is being administered.

*Colitis* is extremely uncommon as the result of the ordinary mercurial course of treatment.

*General malaise* is apt to result from pushing mercury too freely, and it is always advisable to keep a close watch on the patient's weight and general condition.

*Dermatitis* as a result of mercurial treatment alone is very rare, but since arsenobenzol treatment tends to act in the same direction it may be advisable, where there is reason to suspect that the skin is susceptible, to withhold the mercury whilst arsenobenzol is being administered.

**Iodine preparations.**—The rôle of iodine, of promoting the resolution of syphilitic processes, is more particularly required in those stages where the tissue reaction is the greatest, namely the tertiary, but, since the tissue reaction is qualitatively the same in all stages and there are grounds for supposing that this reaction tends to make the spirochaemes inaccessible to antisyphilitic remedies, there is reason for the employment of iodine preparations in all stages. Usually I have given them for short periods between courses of treatment by arsenobenzol and mercury, on the principle of stimulating the tissues to clear away the exudates as rapidly as possible and so to prepare the ground for the further action of the more definitely specific arsenobenzol and mercurial remedies. The favourite preparation is potassium iodide, in doses of 5–30 gr. thrice daily. It may cause gastro-intestinal disturbance, even when given very dilute in water, and is then better tolerated if made up in a cent. per cent. solution and the dose dropped into milk. The depressing effect of potassium iodide is overcome by giving it with nuxvomica, while liquor arsenicalis is believed to counteract its tendency to cause pustular eruptions. When potassium iodide is not well

borne it may be replaced by sodium or ammonium iodide. Many proprietary preparations are advertised as superior to potassium, sodium, and ammonium iodides, but they should be reserved for cases where the older preparations cannot be tolerated. Amongst others are Lipoiodine, sold in tablets; Iodeol, a colloidal preparation given intramuscularly; and collosol iodine given intravenously.

**General management of syphilis.**—The main principles to be observed in the treatment of syphilis are :—

- (1) To begin as early as possible, before the parasite has become buried in the sclerosed primary sore or entrenched in comparatively inaccessible regions, such as the central nervous system.
- (2) To continue as long as experience shows that there is a possibility of the patient relapsing if treatment ceases.
- (3) To exploit the patient's natural resistance, by maintaining his general health in the highest possible condition.

To lay down a line of treatment which will ensure 100 per cent. of cures, yet not err on the side of over-treatment in the majority of cases, is quite impossible. There is evidence to show that, judging by persistence of a negative Wassermann reaction for some years afterwards, and the propagation of healthy children, many cases are cured by a comparatively short course of arsenobenzol and mercurial injections, whilst others, apparently the same in character, have relapsed again and again after the same course. It has become a fairly general custom in early cases to give a course of arsenobenzol and mercurial injections, usually about seven or eight of each, and then, if the Wassermann reaction has become negative, follow up with treatment by mercury for a year or more. It seems to me that too much reliance is placed on this follow-up treatment by mercury, and that in most cases the arsenobenzol and mercurial treatment should be continued for much longer than is usual. Another tendency is to give just enough arsenobenzol and mercurial treatment to convert the Wassermann reaction to negative and then to watch the behaviour of the blood reaction. In such cases another course, and no more, is given when the reaction again becomes positive, in spite of the fact that much more treatment is necessary for a relapse than a fresh case; in this way a patient may



## SYPHILIS

eventually receive far more treatment than would have sufficed to cure him if the courses had followed one another without regard to the blood reaction. I prefer to continue the arsenobenzol treatment far beyond the stage when the Wassermann reaction first becomes negative. Rather than continue with mercury after a minimal course, I would repeat the arsenobenzol and mercurial course.

In all cases of primary sore it is of the greatest importance to apply local treatment to destroy the organisms *in situ*. It may be possible to remove the sore by circumcision, or to destroy it with a cautery. Failing this, the sore may be injected with 0.2 gm. hectine, as supplied in ampoules ready for use. An ointment made up with "914" to 3 per cent. is another useful application, as also is Metchnikoff's cream (p. 312). If an ointment is used, it is a good plan to tell the patient to try to rub the sore away with it. He should be impressed with the fact that rapid healing is not nearly so important as reaching the depths of the sore with the remedy.

The following are the courses which are given at the St. Thomas's Hospital V.D. Treatment Centre to men of average build in different stages of syphilis. For women who are not pregnant it may be advisable to reduce the dosage according to weight.

### ROUTINE TREATMENT OF ADULT MALE SYPHILIS CASES

EMPLOYING AS THE ARSENOBENZOL REMEDY  
NEOSALVARSAN (or one of its substitutes)  
OR SULFARSENOL

#### I. Primary Cases with Negative Wassermann

		Intravenous in- jections of " 914 " or subcutaneous Sulfarsenol	Intramuscular injections of Mercury
	<i>Day of Treatment</i>	Grammes	Grains
1.	1	0.45	i
	8	0.45	i
	15	0.45	i
	29	0.6	i
	36	0.6	i
	50	0.75	i
	57	0.75	i
	58—77	Potassium iodide	
	78	0.75	i
	85	0.75	i
	92	0.75	i
	Blood test on 92nd day		
2.	93—126	Rest	

3. 127-147 Potassium iodide gr. v t.d.s.  
first week, gr. vii second week,  
and gr. x third week
4. 148-239 Repeat treatment as from 1st-  
92nd day
5. Suspend all treatment but test the blood every  
three months.
6. At end of one year test blood after provocative.
7. During second year test blood every six months.
8. At end of second year test blood after pro-  
vocative.

#### II. Primary Cases with Positive Wassermann

Day of Treatment		
1.	1-239	As in I, 1-4. Blood test on 92nd day
2.	240-307	Rest
3.	308-329	Potassium iodide
4.	330	"914" { 0.6 Mercury gr. i
	337	
	344	
	358	
	365	
		or { 0.6 " " i
		Sulfarsenol { 0.75 " " i
		{ 0.75 " " i
5.	Suspend all treatment and continue blood tests at intervals as in I, 5-8.	

N.B.—This programme is based on the assumption that, at the end of the first 10 injections, the Wassermann reaction was negative. If the reaction was positive after the 10th injection, proceed as laid down below for cases with secondary signs.

#### III. Cases with Secondary Clinical Signs

Day of Treatment		
1.	1-365	as in II, 1-4 Blood test on 92nd day and on 365th day
2.	366-433	Rest
3.	434-455	Potassium iodide
4.	462	"914" { 0.6 Mercury gr. i
	469	
	476	
	490	
	497	
		or { 0.6 " " i
		Sulfarsenol { 0.75 " " i
		{ 0.75 " " i
5.	Suspend treatment and continue observations as in I, 5-8.	

#### IV. Tertiary and Latent Cases

Day of Treatment		
1.	1-57	1st course of 7 injections as in I, 1
2.	58-77	Potassium iodide
3.	78-147	Rest
4.	148-182	Course of 5 as in II, 4
5.	183-203	Potassium iodide
6.	204-336	Repeat 78-203
7.	337-469	Repeat 78-203
8.	470-567	Rest
9.	568-602	Course of 5 as in II, 4
10.	603-623	Potassium iodide
11.	624-749	Rest
12.	750-784	Course of 5 as in II, 4

## SYPHILIS

### V. Nerve Cases

Arseno-argenticum or Silver Salvarsan		Intravenous in-
Day of Treatment		jections Grammes
1		0.15
4		0.15
8		0.15
11		0.15
15		0.2
18		0.2
22		0.2
25		0.2
29		0.2
32		0.25
36		0.25
39		0.25
43		0.25
44—50	Pot. iod. gr. v, t.d.s.	
51—57	Pot. iod. gr. x, t.d.s.	
58—64	Pot. iod. gr. xv, t.d.s.	
65—84	Rest	
2nd course. . 85—148	Repeat 1—64	
149—203	Rest	
3rd course. . 204—267	Repeat 1—64	
268—350	Rest	
4th course. . 351—413	Repeat 1—64	
414—511	Rest	
5th course. . 512—575	Repeat 1—64	

**Observation of the patient after suspension of treatment** is of the very greatest importance, especially in early cases; and the longer the period of observation is prolonged, the safer the patient. Before deciding to suspend treatment the blood should be examined and, if practicable, the cerebro-spinal fluid; in the event of either proving to be pathological, the treatment should be renewed after a suitable interval. Subsequently, during the first year, the patient should be examined clinically at intervals of not more than a month, and his blood tested at intervals of not more than three months. At the end of a year from suspension of treatment a small injection of "606" (0.3 grm.) or of "914" (0.45 grm.) should be given, and the blood serum tested, if possible, 2, 4, 7, 13, and 21 days afterwards. If this is not practicable, the seventh day after the provocative injection may be chosen as the most likely to catch a provoked reaction. In addition to the blood test, it is important after a provocative injection to test the cerebro-spinal fluid; this should be done a week after the injection.

During the second year, the periods of

observation may be lengthened, and a six-monthly test should suffice.

**When to discharge the patient.**—The question arises, After what interval of freedom from all signs would one be justified in giving the patient a clean bill of health, and allowing him to marry, for instance? In view of the fact that the arsenical treatment of syphilis and the Wassermann test are of such recent origin, it is impossible to answer such a question with certainty at present. All that can be said is that the best-treated patient should wait for at least two years, and the longer after this that he waits, the better guarantee he has of safety in marriage. As I have said, the risk of transmission is gradually reduced after the second year in any case, and is very uncommon after the fourth year. Judging from the rapid effect of the modern treatment of syphilis on accessible spirochaemes, one would say that a patient could not transmit the disease whilst actually undergoing treatment.

**Pregnant women,** in the early months, may receive the course mentioned above from the 1st to the 57th days, "914" being employed and the dose not exceeding 0.6 grm. On account of the susceptibility of the kidneys, it is advisable to withhold mercury for the first few injections until it is seen that the kidneys are tolerant. Following on this course, potassium iodide is given for two weeks, and the arsenobenzol and mercurial course repeated after two months. On this principle treatment may be continued almost to the end of pregnancy, a careful watch being kept on the kidneys throughout.

**Syphilitic infants.**—The treatment of syphilitic infants is carried out on the same principles as the treatment of adults.

When the mother has been treated throughout pregnancy, it is still advisable to continue treatment of the infant for at least a year. When treatment commences only after birth the child is in a position analogous to that of an untreated adult many months after infection, and its treatment should continue for at least two years.

The best arsenobenzol compound for the purpose is "914," and the dosage is regulated by the body-weight, giving 0.004 grm. per lb. as a commencing dose and working up to 0.007 grm. per lb. at the end of the course. The injections may be given intravenously, into the anterior fontanelle, or a scalp vein, or the external jugular; or intramuscularly into the buttock.

## SYRINGOMYELIA

Mercury is given in the form of hydrargyrum cum creta by the mouth; intramuscularly in doses of  $\frac{1}{4}$ – $\frac{1}{2}$  gr. according to age; or by inunction. In inunction the binder is smeared with cod-liver oil, and a piece of ointment the size of a pea placed under it each morning.

Such a tonic as syrup. ferri iodidi should also be administered. If the mother is under treatment and nursing her infant, the latter benefits from the derivative of arsenobenzol which is excreted into the milk.

L. W. HARRISON.

**SYPHILIS OF NERVOUS SYSTEM** (see CEREBRO-SPINAL SYPHILIS).

**SYRINGOBULBIA** (see SYRINGOMYELIA).

**SYRINGOMYELIA.**—A chronic disease of the spinal cord characterized by the gradual development of a cavity in its substance.

**Pathology.**—The cavity usually extends for a variable distance through the cervical and dorsal regions; it does not, as a rule, spread much into the lumbar segments, although occasionally it is found throughout the whole length of the cord. Sometimes it reaches beyond the cervical region into the brain-stem. It is very variable in its transverse size and outline at different levels, and may branch off in diverticula, or end at one level to begin again at another. It is lined by soft, gelatinous, neuroglial tissue, and its origin is probably due to a process of gliosis in the postcentral part of the cord, associated with some congenital anomaly. An incomplete fusion of the dorsal columns in the midline, with proliferation of embryonic tissue in the walls of the cleft, may form the starting-point in some cases. In others, congenital rests of glial cells near the base of the dorsal columns or in the neighbourhood of the central canal begin to proliferate, and as this growth increases in size it breaks down in the centre, forming a cavity. The occurrence of some cases after injuries to the back has led to a theory that trauma may sometimes act as an exciting cause.

The white and the grey matter at the site of the lesion are destroyed by actual invasion and inclusion in the gliosis, and affected by the compression of the growth and the œdema surrounding it; the progressive involvement of nerve tissue that takes place in this way is responsible for the symptoms of the disease.

**Symptomatology.**—The clinical phenomena—usually beginning gradually in early adult

life, in both men and women—may be described briefly as they occur in the ordinary or cervico-dorsal form of the disease. Motor, sensory, and trophic symptoms are present in varying degrees in different cases.

The **motor disturbances** begin with weakness and wasting in some of the muscles of the upper extremity, usually in the small muscles of one or both hands, but occasionally in the muscles about the shoulder-girdle. The wasting spreads to the forearms, arms, and trunk, but it may long be irregular in its distribution and unequal on the two sides. The hands suffer more than the other parts of the limbs, and tend to assume a "claw-hand" or "preacher's hand" appearance. The wasting very rarely spreads to the lower limbs; more commonly, as the disease advances, a spastic weakness develops in them with the usual signs of that condition, brisk knee-jerks and ankle-jerks and extensor plantar responses. If the disease continues to advance, the patient at length becomes bedridden.

The **sensory symptoms** are very characteristic. The **cutaneous loss** is a dissociated one—that is, tactile, painful, and thermal sensibilities are not all affected to the same extent nor over the same or coterminal areas of skin. Usually there is a greater impairment to moderate degrees of heat and cold than to pain, and sometimes more to warmth than to cold, while tactile sensibility is often normal in areas where the other forms are lost. Thus, a patient may be able to feel light touches on his hands and fingers quite well, and yet experience no pain when he cuts or burns them. On the trunk and legs the loss is frequently crossed, tactile sensibility being affected on one side, and painful and thermal sensibilities on the other.

Impairment of cutaneous sensibility of this kind begins early, usually in the hand or other part of the upper extremity, sometimes on the trunk; later, it involves all these parts more or less; and in the end it is not unusual to find it over almost the whole body.

Combined with the cutaneous loss there may sometimes be impairment of *deep sensibility*, such as the appreciation of position and recognition of passive movement at the joints. Subjective sensations, too, are not infrequent, and the patient may complain of a feeling of numbness, tingling, or burning in the hand or forearm.

**Trophic symptoms** and **vaso-motor disturbances** in the extremities, while not so constant

## TABES DORSALIS

in their occurrence as the motor and sensory conditions, are present in many cases. Painless whitlows, and sores and scars where the patient has burnt or injured himself owing to his anæsthesia, are common on the fingers and hands. Frequently these sores lead to considerable destruction of tissue, and there may be mutilation of the fingers, deformity of the phalanges, or malformation of the nails. The paralysed hand, with its fingers flexed into the palm, often has a soft, oedematous-looking appearance constituting the "succulent hand" of syringomyelia. Occasionally a Charcot's joint is present at the shoulder or elbow, and spontaneous fractures may occur in the bones. A type of the disease in which sensory and trophic disturbances of the hands are prominent and early features has been described as *Morvan's disease*.

Other symptoms that occur are a slight loss of sphincter control, which may develop with the spastic paralysis of the legs; a scoliosis or kyphoscoliosis in the upper dorsal region, which is frequently present and may be of muscular or trophic origin; and a cervical sympathetic syndrome which arises from involvement in the cord of the fibres of the cervical sympathetic, and shows itself in a narrowed palpebral fissure on one side, with a small pupil that does not dilate well to shade, and in a disturbance of sweating or blushing on one side of the face.

If the syringomyelic cavity is present in the upper cervical region or extends into the brain-stem it will cause motor and sensory symptoms at the corresponding level; there

may sometimes be a high-crossed anæsthesia or a spastic condition of the upper extremities. Nystagmus and sensory loss in the area of the fifth nerve are common. The lesion may invade the nuclei of the lower cranial nerves and lead to a wasting of the tongue or a paralysis of the palate or of the laryngeal muscles, or even to a paralysis of one side of the face. The brain-stem form of the disease is known as *syringobulbia*.

The course of the disease is very slowly progressive and it may last for years. Some cases seem to come to a standstill. Sometimes there are periods of sudden increase in the symptoms; cases occurring after injury are apt to progress more quickly than others. Death usually comes from some complication or intercurrent condition, but in syringobulbia may be due to involvement of the medullary centres.

**Diagnosis.**—The disease must be distinguished from various other conditions, but in the ordinary form the syndrome of wasting of the small hand-muscles with dissociated sensory loss occurring in a young adult is very suggestive and, if combined with a cervical sympathetic palsy or with an upper-neurone paralysis of the legs, a scoliosis or trophic disturbances in the hands, is pathognomonic.

**Treatment** is directed to alleviation of the various disabilities. There is no radical treatment, although some observers have thought that X-ray applications over the cervico-dorsal spine have succeeded in arresting the process.

P. W. SAUNDERS.

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**TABES DORSALIS.**—Tabes dorsalis, or locomotor ataxia, is one of the commonest of the chronic diseases of the nervous system. It occurs usually in the middle period of life, and in men much more frequently than in women. Sometimes it is seen about the age of puberty in subjects of congenital syphilis. Occasionally conjugal cases occur.

**Etiology.**—It has long been held that the cause of the disease is syphilis, and this view, based originally on clinical observation of the frequency with which a history of syphilis was found in patients suffering from tabes,

has been very much strengthened by the results of examination of the blood and cerebro-spinal fluid by the Wassermann and other tests; these have served not only as an additional proof of the presence of antecedent syphilis in tabetic patients, but as a demonstration of the persistence and continued activity of the virus of syphilis. In one or two cases the *Spirochaeta pallidum* has been found in the spinal cord. The tabetic symptoms do not usually make their appearance until long after the occurrence of the syphilis. The interval between the two conditions may

## TABES DORSALIS

be anything from four to twenty-five years or longer, but is usually about ten or twelve years.

Other causes besides syphilis that have been thought to play a part in producing the disease are strain and stress, excessive fatigue, over-exertion, alcoholic and sexual excesses, and trauma. It is difficult to see how these can act, except as contributory causes. Sometimes, for instance, the symptoms date from an accident or a fall. In many such cases, however, examination after the accident reveals such advanced signs of the disease that it must be held that the condition was in existence long before the accident, and that the latter only precipitated the onset of the symptoms.

**Pathology.**—The pathological changes involve especially the sensory tracts in the dorsal columns of the spinal cord. These show a progressive degeneration which usually begins in the lower part of the cord. In an early case it is found in the lumbo-sacral region, in the outer part of the column of Burdach; a little later it involves also the column of Goll, and may be traced upwards into the dorsal and the cervical regions. As it is followed up the cord it occupies less and less transverse extent and moves towards the middle line, so that in the upper dorsal and cervical regions it may be confined entirely to the column of Goll. In the later stages, however, the whole transverse area of the dorsal columns throughout the length of the cord may be involved to a greater or less extent, although even in the most advanced cases certain small endogenous tracts, the septo-marginal, the cornu-commissural, and the comma tracts, are usually spared. As the fibres disappear from the degenerated areas a secondary neuroglial proliferation takes place. Occasionally the chief early incidence of the degeneration is on the cervical region, and the first lesions implicate the columns of Burdach in this region.

The origin of the degeneration has been the subject of much discussion. It is the exogenous fibres of the columns that suffer—those, namely, whose cells are in the ganglia on the posterior roots—but no changes have been found in the ganglion cells that would account for the degeneration. According to one hypothesis, it results from a syphilitic meningitis that involves the posterior surface of the cord and constricts the dorsal roots at their point of entry. As many cases show an infiltration and thickening of the pia mater around the

posterior roots, this explanation has considerable evidence in its favour.

There is reason, too, for thinking that in tabes the path of infection may be by way of the perineural lymphatics of the afferent nerves, and that the nerve-fibres are especially vulnerable to the virus at their point of entrance into the cord, where they lose their neurilemmal sheaths. It is at this point that the degeneration begins. These considerations support the theory that tabes may be due to a "lesion of the lymph system of the posterior columns, the membranes covering them, and the posterior roots" (McIntosh and Fildes). But the exact pathogenesis of the disease is not yet settled.

In addition to the dorsal columns, Lissauer's zone frequently degenerates, and occasionally there are degenerative changes in some of the cells in the anterior horns. As the disease advances, the extramedullary portions of the roots and the cells of the spinal ganglia atrophy, and the peripheral nerves, especially the sensory nerves in the skin, may show a variable amount of degeneration.

The disease is not limited to the spinal cord, as the optic nerves often undergo a primary atrophy, and the third, fourth, and sixth nerves may be involved; lesions of the auditory, the vagus, the trigeminal, and the hypoglossal nerves are rarer.

The cerebro-spinal fluid frequently contains an excess of lymphocytes and a slight increase of albumin, and it usually gives a positive Wassermann reaction. The Wassermann test is commonly but less constantly positive in the blood also; occasionally it is negative in both blood and fluid.

**Symptomatology.**—The symptoms of tabes are very numerous, and vary considerably in different cases. It will be well, therefore, to describe the more important of them briefly first, and then discuss their association in the different types of the disease.

Subjective **sensory disturbances**, such as feelings of tingling or numbness in the feet or hands, are very common, or the feet may feel swollen and large and the soles numb and dead as if the patient were walking on sand or a thick carpet. Or there may be feelings of constriction about the big toe as if the skin were too tight, or about the knees, or a girdle sensation about the body as if something were tied tightly round it. Sudden acute stabs of pain—the so-called lightning pains—occur most commonly in the lower extremities, but also

## TABES DORSALIS

in the trunk or the upper limbs. Dull aching pains are felt in the joints and elsewhere. These paræsthesiæ and pains are usually among the early symptoms, but may last long into the course of the disease and be present at any stage.

Objective sensory loss affects both the cutaneous and the deep forms of sensibility. The cutaneous loss may involve all forms of cutaneous sensibility, but it is not uncommon for it to be dissociated, appreciation of pain or of temperature being more affected than tactile sensibility. Sometimes the analgesia in the legs is accompanied by an over-response to painful stimuli that the patient feels, or appreciation of the pain may be delayed. Areas of hyperæsthesia may occur also, especially on the trunk.

The cutaneous sensory affection usually begins at the distal parts of the legs and spreads higher as the disease advances, frequently till it reaches to about the level of the nipples. Sometimes it begins on the trunk in the form of a band around the chest at this level, or in scattered patches on the body. On the upper extremities it commonly involves first the ulnar side of the hand and forearm.

More characteristic than the cutaneous disturbance is loss of the sense of position, of recognition of passive movement, of deep-pressure pain, and of appreciation of vibration. These are forms of sensibility conducted in the long fibres of the dorsal columns; their disturbance begins in the feet and lower part of the legs and extends later to other parts of the lower limbs, and still later to the upper extremities. Owing to this sensory trouble the patient is unable to know the position of his feet or of his legs without seeing them. Therefore his gait becomes unsteady, he shows *Romberg's sign* or sways when he stands with his feet together and his eyes shut, he is unable to get about in the dark, he walks on a wide base, lifting his feet too high, putting them down clumsily and watching the floor as he places them; and if the condition progresses he becomes at length so grossly ataxic that he cannot walk at all. When his hands are involved he cannot use them without watching constantly what he is doing, fine movements become impossible, and he cannot even keep his outstretched hand still without looking at it.

Compared with the sensory symptoms, purely **motor symptoms** are much less prominent. The muscles become flabby and hypotonic,

especially in the lower extremities, and this leads to an abnormal freedom of motility at the different joints. In rare cases localized wastings of muscles occur in the lower or upper limbs.

The deep **reflexes** diminish gradually and finally disappear. The ankle-jerk and the knee-jerk, the first to be affected, are lost comparatively early in the disease. The arm-jerks may be affected or not, according to the extent of involvement of the upper part of the cord. The skin reflexes and plantar responses are not necessarily altered.

**Sphincter troubles** are commonly present from the early stages: they vary in prominence and severity in different cases, and may be subject to periods of improvement or exacerbation. The bladder sphincter is usually more affected than the rectal, and there may be a dysuria, a frequency, or an incontinence of urine. In many cases there is poor rectal control after the taking of aperients. Sexual impotence also may be among the early symptoms, or may develop as the disease progresses.

The **ocular symptoms** of the disease are of very great importance. The pupils, which are usually small, may be unequal, irregular in outline or eccentric in situation, and sluggish or inactive to light while still reacting on accommodation. This reflex inactivity is the *Argyll-Robertson phenomenon*. There is frequently, too, a drooping of the eyelids, often unequal on the two sides, and this, with the small pupils, gives the patient a very characteristic facies. Often there are strabismus and diplopia. Primary optic atrophy is of not infrequent occurrence also, usually leading to more or less complete blindness.

Among the less constant manifestations of the disease are the **visceral symptoms**, paroxysmal attacks of pain and distress involving different viscera and usually described as *crises*. The most common are gastric crises, with attacks of uncontrollable pain and vomiting, which may persist for several days, leaving the patient exhausted (*see* Gastric Crises of *Tabes*, under **STOMACH, FUNCTIONAL DISORDERS** or): rectal crises, characterized by straining and tenesmus; bladder crises, with constant desire to micturate and inability to do so; and laryngeal crises, in which the patient gets attacks of coughing associated with spasm of the laryngeal muscles and the glottis.

In certain cases **trophic symptoms** occur. Mention has already been made of the localized muscular wastings. Arthropathies, or

## TABES DORSALIS

Charcot's joints, are very characteristic of the disease. Usually they lead to much enlargement of the joint, with accumulation in it of fluid, disintegration of the ends of the bones, osteophytic outgrowths, and more or less articular disorganization. They occur most commonly in the lower extremity. Among other trophic disturbances are rarefaction of the long bones which may lead to spontaneous fractures, and deep perforating ulcers on the soles of the feet.

**Stages.**—The ordinary type of the disease is very gradual in onset, and is usually described in three stages. There is first a *preataxic stage* of very variable duration, characterized by the presence of subjective sensory symptoms, such as numbness and tingling in the feet and fingers, vague wandering pains about the body and lightning pains in the legs or trunk or arms, which are often ascribed by the patient to rheumatism. Ocular symptoms may be present also, and some sphincter trouble, or some of the less frequent symptoms already mentioned. On examination the patient usually presents unequal or irregular pupils which are sluggish or inactive to light, slight drooping of the eyelids, diminution or loss of one or both ankle-jerks or knee-jerks, and sensory loss in the lower extremities. In the next or *ataxic stage* this sensory loss becomes greater, there is an unsteadiness of gait and a difficulty in getting about in the dark, while the pains and sphincter trouble and other symptoms continue or get worse. Gradually the ataxia and the hypotonia which accompanies it become greater, and the patient after a variable period passes finally into the *paralytic stage*, in which, owing to loss of the sense of position in the larger joints, he is wildly ataxic and bed-ridden from inability to co-ordinate his movements. But this division into stages is misleading, for in many cases the disease does not present the sequence of events that such a division suggests, and many patients never pass through all these stages.

**Types.**—*Cervical tabes*, in which clumsiness in the hands and pains in the upper extremities are among the initial symptoms, is much less common than the ordinary type just described, in which the lower extremities are chiefly affected. The cervical cases are those in which the disease affects first the cervical enlargement instead of the lumbar.

In another variety of the disease the symptoms for which the patient seeks advice are *ocular*. He may have ptosis or an ocular

palsy, or may complain of failing vision. On examination, some sensory loss may be discovered in his legs with diminution or absence of knee-jerks or ankle-jerks, and perhaps sphincter trouble, and even reflex changes may be absent.

In the *neuralgic type* lightning pains and other subjective sensory disturbances are the most prominent features of the disease; they may occur in bouts lasting several days at a time. In other types the sphincter trouble is the predominant feature; or gastric or other visceral crises may be the symptoms for which the patient seeks relief. Their nature may at first be obscure unless some of the other signs of tabes, such as pupillary or reflex changes or sensory loss, be present. In other cases trophic disturbances, an arthropathy, or a perforating ulcer, may be the most noticeable feature, and the ordinary classical symptoms may be in abeyance.

There is a form of the disease in which the patient is depressed and nervous about himself, complains of headaches and giddiness, does not sleep, cannot concentrate his attention, and is full of phobias, and especially syphilophobia. He may have vague indefinite pains, or even shooting pains, and perhaps some doubtful double vision. As the ordinary signs of the disease are late in appearing, the case may seem for a time to be merely one of neurasthenia.

Mental symptoms are not common, and the mind remains quite clear till the end. Sometimes, however—usually after the disease has been in existence for some time—the mental phenomena of general paralysis of the insane develop. These cases of tabo-paresis represent an overlapping of the two diseases; the patients have the spinal symptoms of tabes and the cerebral symptoms of general paralysis.

*Congenital* or *juvenile tabes* does not differ materially from the ordinary forms just described, except that it is more apt to be of the tabo-paretic variety, and optic atrophy and deafness are not uncommon features.

The **progress of the disease** is very variable in different cases. Some may last for years without advancing, or may advance very slowly. Frequently there are periods during which there is considerable increase of the symptoms, with intervening periods in which they are more or less stationary. The pains usually tend to get less constant and severe, although they sometimes last far on in the course of the disease. The same applies to the crises with

## TABES DORSALIS

which the pains are often associated. Patients in whom the disease begins with special features like crises or trophic disturbances may never develop much ataxia. Especially is this true of the cases that begin with optic atrophy, which may remain throughout the principal feature.

**Diagnosis.** — The diagnostic features of chief importance are the lightning pains, Argyll-Robertson pupil, absent or diminished knee-jerks and ankle-jerks, Romberg's sign, the characteristic sensory loss, and the sphincter disturbances; positive Wassermann reactions in the blood and cerebro-spinal fluid often help to confirm the diagnosis. When the disease is advanced its diagnosis is easy, but in the earlier stages, before the physical signs become definitely established, it is sometimes difficult to come to a conclusive diagnosis straight away, and in such cases the examination of the blood and cerebro-spinal fluid may be of great help.

The visceral crises, especially if they occur early, may suggest primary disease of the different viscera, and it may be difficult to exclude this unless the other signs of tabes are sought for very carefully. Sometimes, too, in cases of *neurasthenia* of the depressive variety, associated with insomnia, arteriosclerosis, and even syphilophobia, early tabes may be missed until the characteristic pupil and reflex changes develop.

*Peripheral neuritis* may simulate tabes very closely, for there may be pains, loss of power and ataxia in the legs, disappearance of the deep reflexes, and occasionally sluggishness of the pupils. But in neuritis the upper extremities are apt to be involved equally with the lower, there is actual loss of motor power, the sensory affection differs from that of tabes in its distribution and character, and the muscles are hypersensitive to pressure, while in tabes they are generally insensitive. Sphincter disturbances are less common unless there is associated mental disturbance, and the peculiar form of mental dissociation that occurs in alcoholic neuritis is quite unknown in tabes.

Lesions affecting the dorsal columns may give rise to symptoms resembling those of tabes particularly a *meningo-myelitis* involving especially the dorsal surface of the cord. The differences in onset and progress, the absence of the pupillary phenomena and lightning pains, and the results of the Wassermann test are of importance in the diagnosis.

*Subacute combined degeneration of the spinal*

*cord* may involve the dorsal columns much more than the lateral, and lead to a condition of flaccid ataxic paraplegia with absent deep reflexes, sensory disturbances, and sphincter trouble. But lightning pains are less common, and the absence of the pupillary changes, the presence of extensor plantar responses, and the results of the Wassermann test are of help in the diagnosis. Similarly, the ataxia and absent deep reflexes of *Friedreich's disease* may suggest tabes, but the differences between the two are so numerous and definite that the diagnosis is readily made, except in atypical cases.

*Chronic cerebellar lesions* in which optic atrophy and ataxia are present may resemble tabes, but the absence of the characteristic pains and sensory disturbances, the type of the ataxia, and other differences in the history and symptoms make the diagnosis possible. Similar considerations apply to tumours and lesions in other parts of the brain which have led to internal hydrocephalus, optic atrophy, absent deep reflexes, and unsteady gait.

The condition of tabo-paresis to which reference has already been made represents the impossibility sometimes of drawing a sharp line between tabes and *general paralysis of the insane*. Similarly, it may at times be very difficult to distinguish clinically these two conditions from certain cases of *cerebral and cerebro-spinal syphilis*, especially as syphilitic meningitis and myelitis may complicate and mask the spinal symptoms of tabes, and disseminated specific lesions may produce symptoms resembling those of tabo-paresis and general paralysis.

**Treatment.** — The association of tabes with syphilis suggests the advisability of employing antisyphilitic measures in its treatment. Various forms of specific treatment have been used, such as the administration of mercury by the mouth over long periods in the form of liquor hydrargyri perchloridi or some of the mercurous salts, the exhibition of the iodides, and the use of mercury by inunction and intramuscular injection. More intensive methods of treatment with salvarsan and similar preparations have also been employed, while intrathecal injections of salvarsanized serum, mercurial serum, and salvarsan have been tried. A very usual method of treatment now is the intravenous injection of the salvarsan drugs combined with intramuscular injections of mercury in courses repeated at intervals of some months.



## TABES DORSALIS

But even the more intensive antisyphilitic treatment does not yield the same beneficial results in tabes as in ordinary primary and secondary syphilis. The lesions in tabes affect parenchymatous nervous tissue in which the power of regeneration is very poor, and once the degenerative process is started in any neurone it probably continues; further, the remedies do not seem to penetrate to any extent into the central nervous tissues. On the other hand, it is claimed that antisyphilitic treatment may modify the virus that causes the degeneration and arrest or lessen the progress of the disease, although it may not effect a restoration of tissue already damaged; and also that not uncommonly meningeal and vascular lesions which are associated with the degenerative changes are amenable to cure and relief by antisyphilitic measures. In actual experience the effects of this kind of treatment vary considerably. In many longstanding cases it produces no improvement whatever, but in some cases—possibly cases in which the condition is less advanced or has followed more quickly after the primary syphilitic infection, or cases with meningeal or inflammatory complications—some of the symptoms seem to be very definitely improved and the general sense of well-being and hopefulness much increased. This is especially so after the injection treatment.

Apart from antisyphilitic measures, tonic and symptomatic treatment is necessary, with plenty of rest and nourishing food. The pains, bladder trouble, constipation, loss of appetite, and general weakness and wasting which are so frequently complained of require special measures. The bladder trouble may be helped by the use of urotropine, atropine or belladonna, or ergot. The crises may necessitate various sedatives, and even opium or morphine. The pains require the use of analgesic drugs, of which the coal-tar preparations and acetylsalicylic acid are the most important. Morphine is better avoided. The optic atrophy has been treated by increasing doses of strychnine and applications of galvanic electricity to the temples, but usually it does not respond to any treatment. The Charcot's joints may require the use of splints or supports of different kinds, while the hypotonia and wasting of the limbs may be benefited by massage and electrical stimulation.

Much may be done for the patient by re-educating him in the use of his limbs by means of graduated exercises on the Frenkel system.

## TABES MESENTERICA

These must be done carefully and persistently, and with a great deal of voluntary attention on the part of the patient, and each treatment should stop short of causing fatigue. The perfunctory carrying out of the exercises does no good. Every attempt should be made to prevent the patient from giving way to despondency and abandonment of voluntary effort.

P. W. SAUNDERS.

**TABES MESENTERICA** (*syn.* Tuberculous Adenitis of Mesenteric Glands).—It is difficult, clinically, to draw a hard-and-fast line between this disease and tuberculous peritonitis, for when symptoms are caused some degree of peritonitis is usually present.

**Etiology.**—Children are especially liable, and the infection would appear to be commonly bovine and derived from tuberculous milk. Local ulceration of the neighbouring bowel is often found, but it has been shown that the bacilli can traverse a mucous membrane which is histologically sound. The possibility of the infection being blood-borne in certain cases cannot be excluded.

**Symptoms** may be altogether absent, or there may be such signs of general ill-health as anorexia, malaise, wasting or failing to gain weight, dyspepsia, disturbance of sleep, and slight fever. In many instances the disease progresses to fibro-caseous or calcareous change without arousing suspicion of its presence or greatly influencing health. In others, abdominal pain, which may be severe and often follows meals, may be complained of. Bouts of diarrhoea or the passage of bulky, undigested stools, perhaps accompanied by mucus and more rarely by blood, may indicate a local disturbance, or there may be constipation. The abdominal wall is usually flaccid, but there may be resistance over the glands. These are situated most commonly in the right iliac fossa or about the umbilicus. Often no glands can be distinguished, but fullness merely; repeated examinations may make the position more plain.

**Diagnosis.**—*Fæcal masses* may be indistinguishable except by free purgation or an enema. Sometimes the illness is more severe, and a definitely tender mass, accompanied by much rigidity, is present in the right iliac fossa and is associated with vomiting. Under these circumstances laparotomy may be necessary to exclude *acute appendicitis*. In less acute cases *chronic appendicitis* provides a difficulty, for it may be impossible by palpation to decide

## TALIPES

whether a small tumour in the right iliac fossa or near the pelvic brim is a gland or a thickened appendix, while the previous symptoms may be alike and may include colic in each disease. *Intestinal obstruction* may be caused by a mass of actively tuberculous glands and the surrounding inflammation, or by strangulation by a resulting fibrous band; the obstruction may be ascribed wrongly to other causes, but this is of little moment, since exploration is necessary. *Renal diseases*, such as calculus, pyelitis, tuberculosis, or tumour may be confused. In renal swellings the situation and the relation of the colonic resonance are important; the urine should be examined in all suspected cases. Rectal examination should never be omitted, and may settle the diagnosis both by rendering the glands palpable and by excluding the other disorders. Radiography may reveal the presence of a calcareous gland, but in some cases ureteral catheterization or pyelography may be necessary to determine that the shadow is not that of a renal or ureteral stone.

**Treatment** is considered under LYMPHATIC GLANDS, TUBERCULOSIS OF. Special attention should be paid to the diet, foods which leave a bulky residue being avoided. Uncooked fruits and vegetables are deleterious, and milk may be ill-borne and should not in any case be given in large quantity. Fats such as cream and cod-liver oil, though valuable, may upset digestion, and a careful watch must be kept on the stools. Surgical treatment is imperative with obstruction, and may be advisable in cases which fail to benefit otherwise; it is usually unnecessary, and not without the risk of sinus-formation or generalized tuberculosis.

FREDERICK LANGMEAD.

**TACHYCARDIA** (see AURICULAR FIBRILLATION; AURICULAR FLUTTER; HEART, IRRITABLE; HEART, PALPITATION OF; HEART-BEAT, ABNORMALITIES OF).

**TACHYCARDIA, PAROXYSMAL** (see PAROXYSMAL TACHYCARDIA).

**TÆNIÆ** (see INTESTINAL WORMS).

**TALIPES.**—A deformity of the foot which occurs chiefly at the ankle and tarsal joints. *Talipes equinus* signifies a foot in which normal dorsiflexion of the ankle is limited and the patient walks unduly on the toes. *Talipes calcaneus* indicates a foot in which plantar flexion is limited and the patient walks on the heel. *Talipes varus* and *talipes valgus* denote feet which are respectively inverted or everted

chiefly at the midtarsal joints. Sometimes there is a complex deformity: thus *talipes equino-varus* and *talipes calcaneo-valgus* are common varieties.

*Talipes cavus* or *pes cavus* signifies a highly-arched foot with contracture of the soft parts of the sole; this is frequently secondary to *talipes equinus*.

**Etiology.**—The causes which lead to talipes are various. Many cases are congenital, some result from infantile or spastic paralysis or peripheral nerve lesions, a few follow trauma, while in rare instances unequal growth of the bones of the leg may produce the deformity. The majority of cases which come for treatment are either congenital or paralytic in origin.

**Congenital Talipes Equino-varus.**—This deformity is one of the commonest seen in an orthopaedic clinic. At birth the foot is healthy, with good circulation and well-developed muscles, but there is inversion at the midtarsal joint and plantar flexion at the ankle. The long flexors and the tibiales anticus and posticus muscles are displaced to the inner side of the foot; even the bones of the tarsus share something of the general distortion of the foot. If the condition does not obtain proper treatment the bones take on a permanently bad position and shape, and the weight of the body is transmitted by the outer border and dorsum of the foot.

**Diagnosis** presents no difficulty, since the condition dates from birth.

**Treatment.**—There are two aims in treatment. First the deformity must be corrected, secondly the corrected position must be maintained by apparatus. The methods of correction vary according to the age at which treatment is started and the degree of bony deformity which exists. Whatever procedure be adopted, some form of retentive apparatus will be required until the foot is fully developed and the deformity no longer tends to recur. The methods of correction are as follows:

**Manipulation.**—This should be started as soon after birth as possible. The foot must be everted at the midtarsal joint and dorsiflexed at the ankle. It is important to grasp the lower epiphyseal region of the leg firmly with one hand while the other hand performs the forcible movement, otherwise damage may be done to the cartilaginous epiphysis. These manipulations should be carried out twice a day for five or ten minutes, and the improved

## TALIPES

position maintained either by a malleable splint (such as Robert Jones's) or a simple rectangular splint with an internal ledge to prevent inversion.

**Forcible wrenching.**—When simple manipulation fails to correct, an anæsthetic must be given and the foot forcibly wrenched into position either by a Thomas wrench or by leverage over a wooden wedge. The soft parts can often be heard to give way as the force is applied. Over-correction is to be aimed at. The foot may be kept in the new position by a malleable splint or by a plaster-of-paris casing applied immediately. The drawback to a plaster splint is that it cannot be removed daily to permit of massage, consequently the limb may atrophy and lose power.

**Tenotomy.**—If manipulation and wrenching are unsuccessful, and in cases in which early treatment has been neglected, it is necessary to divide the tendons of the *tibiales anticus* and *posticus* and the *tendo Achillis*. The plantar fascia also needs division. These are cut subcutaneously by a small sharp-pointed tenotome. After tenotomy the foot is wrenched into an over-corrected position and splinted. Some elect to defer the division of the *tendo Achillis* till after the varus deformity has been fully corrected. Strict aseptic precaution should be observed in the simple operation of tenotomy. The tendon of the anterior tibial can be felt subcutaneously on the inner aspect of the dorsum of the foot, while the posterior tibial tendon is best divided as it lies close against the bone at the lower end of the tibia.

**Open division of the soft structures.**—If simple tenotomy does not allow of full correction, it is necessary either to divide the contracted soft parts by open incision, or to perform some operation on the bony tarsus. Phelps's operation is the one usually performed on the soft parts. Though the immediate result is good, great damage is done to the musculature of the foot, and many surgeons think it an unwise procedure. The incision for this operation extends from just below the internal malleolus to the middle of the sole of the foot. It is carried down to the bones and the astragalo-scapoid joint is opened; any bleeding is stopped, a dressing applied and the foot put in a correct position and maintained so by a plaster splint with a trap-door opening through which the wound can be dressed.

**Operations on the bones.**—In cases where the bones are an obvious obstacle to proper correction it is necessary to remove a section of

the tarsus. Surgeons differ as to the operation of choice. Wedge-shaped resection of the neck of the astragalus, excision of the astragalus, removal of the head and neck of the astragalus together with the anterior portion of the os calcis, and wedge-shaped resection of the outer part of the tarsus, have each their advocates. In some cases simple osteotomy of the astragalo-neck may suffice for correction.

In any case, the minimum of bone which will allow of correction must be removed, and the foot splinted.

**Retentive apparatus.**—In slight cases treated from birth it is usually sufficient to apply a malleable iron, tin, or similar pliable splint (e.g. Jones's) which is sufficiently strong to maintain the correct position and yet can be bent suitably by the operator. If the condition be apparently cured it is still necessary to keep the child under observation, and the outer border of the boot must be raised half an inch to avoid the constant tendency to recurrence.

More stubborn cases need a corrective appliance when the child walks. A sole-plate placed in the shoe and affixed to an upright iron on the inner side of the leg, with a calf-band at the upper end, may suffice. The adoption of the equinus position must be prevented by a stop at the ankle. The Adams shoe is a more elaborate but satisfactory apparatus.

Very occasionally a case of talipes may come to the surgeon in such a neglected condition and advanced state of deformity that the patient would seem better off with an artificial foot, but it is very seldom that amputation is justifiable.

**Talipes Equinus.**—This deformity is sometimes congenital, but may occur in spastic conditions and is sometimes compensatory to a shortened leg, or secondary to drop-foot. It is treated by tenotomy or lengthening of the *tendo Achillis*. The heel of the boot should be low, and the correct position may be furthered by placing a transverse bar of leather across the sole at the tread.

**Pes Cavus.**—Claw-foot is sometimes associated with talipes equinus, to which it may be secondary. Occasionally it is the result of cerebral spastic paraplegia. The arch of the foot is raised and the toes are dorsiflexed, while the heads of the metatarsals are depressed.

**Treatment** in slight cases is by lengthening of the *tendo Achillis*, tenotomy of the plantar fascia, and wrenching. In worse cases division of the tendon of the *extensor longus hallucis*,

## TALIPES

and its fixation to the plantar aspect of the first metatarsal after passing it through the head of that bone, is advisable. Resection of the head and neck of the astragalus has been recommended (Rowlands).

**Talipes Calcaneus.**—This form of talipes requires treatment by manipulation and splinting. It is rarely necessary to perform tenotomy.

**Paralytic Talipes.**—The exact form of club-foot resulting from infantile palsy depends upon the group of muscles paralysed. If the tibial-peroneal group is affected, talipes *equino-varus* results. If the calf muscles are paralysed, talipes *calcaneo-valgus* is the consequence. These are the common forms.

The **diagnosis** of paralytic talipes is easy, since the limb is cold and bluish, the circulation in the foot poor, and the muscles weak or paralysed, failing to react to faradism.

**Treatment.**—In any case of infantile palsy it is most important to maintain the limb in a position which will prevent contracture or stretching of the muscles. At the same time, every encouragement to the recovery of the paralysed muscles must be given, the limb put on a suitable splint, and massage and electrical treatment undertaken.

When the acute symptoms have subsided no operation should be undertaken until all the recovery that is likely to take place has occurred. When no further recovery can be anticipated, three methods of treatment present themselves for consideration: (1) The transplantation of tendons of healthy muscles to take the place of or to reinforce weak or paralysed muscles. (2) Nerve-anastomosis so as to bring living axis-cylinders into the degenerated peripheral nerve supplying the palsied muscle. (3) In cases where all the muscles round the ankle are weak, arthrodesis of the ankle may be advisable.

In every case a boot with supporting iron will be necessary. The iron should fit into a socket in the boot and should reach well up the leg, where it is fixed by a calf-band. The iron should be on that side of the foot to which it tends to be displaced, i.e. the inner side in varus cases, the outer side in valgus cases. A T-strap is fixed to the opposite side of the boot and buckled round the upright iron. If the thigh muscles also are weak a calliper splint with purchase from the hip may need to be worn.

ZACHARY COPE.

**TAPEWORM** (see **INTESTINAL WORMS**).

## TENDONS, WOUNDS OF

**TAPPING** (see **PARACENTESIS**).

**TARSAL CYST** (see **EYELIDS, AFFECTIONS OF**).

**TASTE, DISTURBANCES OF.**—Gustatory impressions from the anterior two-thirds of the tongue are conveyed by the lingual and chorda tympani nerves to the geniculate ganglion, and thence by the nervus intermedius of Wrisberg; while those from the posterior third of the tongue and from the palate and fauces pass through the glosso-pharyngeal nerves. These two sets of taste fibres have probably a common termination in the medulla, near the oral end of the fasciculus solitarius, in the so-called nucleus gustatorius. Taste is affected in the corresponding region when one of these nerves is injured. This may be due to bulbar lesions that involve the dorsal and lateral part of the medulla, or to damage of their roots by such lesions as syphilitic meningitis. But the most common cause of loss of taste is injury to the chorda tympani fibres by a lesion of the facial nerve in the Fallopian canal; the loss is then associated with facial paralysis, and is limited to the anterior two-thirds of the same side of the tongue. This may also result from surgical or other injuries to the lingual nerve. A loss of tactile sensibility on the tongue due to trigeminal anaesthesia may blunt or even abolish taste in the same region.

There is often a diminution of taste in such diseases as tabes and general paralysis, and various perversions occasionally occur in the functional neuroses. Cerebral disease never abolishes the sense of taste, but irritative lesions on the inner surface of the temporal lobe may excite transient gustatory auræ, generally of an unpleasant nature.

GORDON HOLMES.

**TEETH, AFFECTIONS OF** (see **DENTAL CARIES**; **DENTAL, DENTIGEROUS AND MULTILOCULAR CYSTS**; **DENTAL PAIN**; **DENTITION AND DENTAL IRREGULARITIES**).

**TEMPORO - SPHENOIDAL LOBE, LESIONS OF** (see **NERVOUS SYSTEM, CENTRAL, LOCAL LESIONS OF**; **CEREBRAL TUMOUR**).

**TENDONS, WOUNDS OF.**—Usually it is at the wrist that these wounds occur, the hand being thrust through a pane of glass; or the slipping of a knife severs the tendons of a finger or thumb. Very often there is an associated injury to a nerve or large blood-vessel.

## TENDONS, WOUNDS OF

**Treatment.**—Any serious hæmorrhage having been stopped temporarily, it is essential by testing function to ascertain, before anæsthesia is induced, which tendons are cut, and whether there is any nerve lesion. A longitudinal incision should be made over the injured tendons, the surrounding skin and the accidental wound itself having been cleansed previously by the accepted methods of wound treatment. The distal end of the tendon can be made to protrude by flexion or extension of the fingers or wrist, according as the wound is on the anterior or the posterior aspect of the hand. The proximal end retracts further; it should not be sought for by thrusting sinus forceps up the sheath, but by sufficient exposure. By pressing upon the attached muscle-bellies it is possible to squeeze out the tendons for some distance; when they appear in the wound they are grasped with forceps. If many tendons are divided, great care must be taken to suture the corresponding ends in apposition. The **method of suture** is important. Chromicized catgut is used. There are two practical methods of suture:

(a) *For small flat tendons.*—The cut ends are made to overlap slightly and kept in apposition by two mattress sutures (Fig. 93).

(b) *For larger tendons.*—A ligature is tied tightly round the tendons to be approximated,

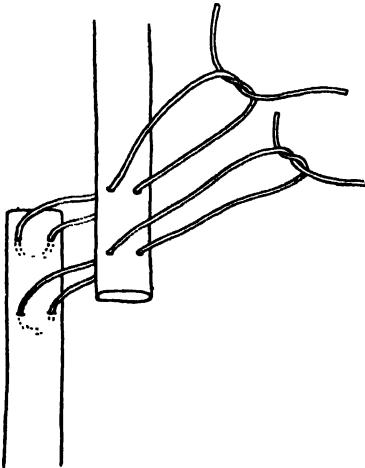


Fig. 93.—Method of suturing flat tendons.

$\frac{1}{8}$  in. from their divided ends. This holds the extremities of the parallel-running fibres together and prevents the connecting stitches from pulling through. These latter are two in number and placed through the tendons in

## TENESMUS

two planes at right angles to each other (Fig. 94).

When possible, the tendon sheath should be sutured with fine catgut. It is important to suture the deep fascia overlying the junction, especially if it has been divided where it forms

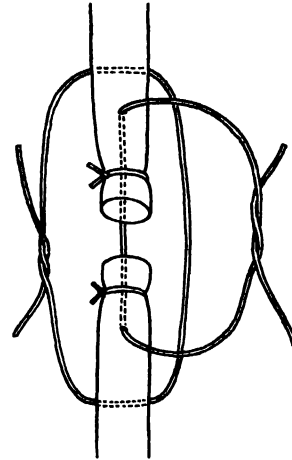


Fig. 94.—Method of suturing cylindrical tendons.

the anterior or posterior annular ligaments. To prevent adhesions to skin the fatty superficial fascia must be stitched separately. The limb is placed so that the sutured tendons are fully relaxed. Gentle, small-range, passive movements should be practised on the eighth day, when the skin sutures are removed, but earlier than this slight active movement should be encouraged. No severe strain should be put upon the new junction for three weeks, and this means the maintenance of the position of relaxation for that period; but movements must be carried out daily. With such careful after-treatment the subsequent breaking down of adhesions under anæsthesia will seldom be called for.

C. A. PANNETT.

**TENESMUS.**—Continued uncontrollable painful efforts to empty the bladder or the rectum. *Vesical* tenesmus is common when the bladder, in the region of its neck, or the prostatic urethra is inflamed; thus it occurs in cystitis and in gonorrhœal posterior urethritis or prostatitis. A very painful and intractable form is met with in carcinoma of the bladder, and in carcinoma of the prostate which has invaded the bladder-wall. Tenesmus may be due also to retention of urine whether by stricture, prostate, or stone. Reflexly, too,

## TENO-SYNOVITIS

vesical tenesmus occurs during renal colic, and it may be accompanied by rectal tenesmus. *Rectal tenesmus* is usually associated with simple proctitis or that due to piles, polypus, new growth, fissure, or a fistula. A foreign body in the rectum will also cause it.

In the **treatment** of tenesmus the underlying cause should be attacked, and for this the appropriate article should be consulted. The painful contractions, whether of the bladder or of the rectum, may be relieved by the application of heat and local sedatives. Fomentations are applied to the bladder or perineum, or the patient may sit in a hot bath. Into the rectum should be inserted a suppository composed of belladonna extract 1 gr., morphine  $\frac{1}{4}$  gr., theobroma oil q.s.; or a small enema (2 oz.) of starch mucilage to which 20 min. of liq. opii have been added may be administered. The tenesmus associated with malignant disease of the bladder or rectum is controlled with difficulty, even by large doses of morphia subcutaneously. Colostomy or permanent suprapubic drainage will give relief for a time.

C. A. PANNETT.

**TENO-SYNOVITIS.**—Inflammation of the synovial sheath surrounding a tendon.

**Etiology.**—The condition may be due to (a) over-use or strain of the attached muscles, or to (b) bacterial infection, whether directly implanted as in septic wounds, or carried by the blood-stream as happens in the case of the gonococcus and the tubercle bacillus. Teno-synovitis may also be caused by syphilis and by rheumatism.

**Varieties.** Teno-synovitis may be acute, subacute, or chronic. Septic organisms and the gonococcus cause an acute, the tubercle bacillus a chronic form, while the lesion following over-use is subacute.

**Pathology.**—In the *acute* type the inflammatory changes in the lining endothelium cause effusion of fluid, sometimes purulent, and, later, extensive adhesions between the walls of the sheath, leading to great disability in moving the muscles. There is perisynovial extension of the inflammation. In the *subacute* type there is a change in the endothelium (probably leading to diminution of fluid secretion) which causes the characteristic crepitus. In *chronic* cases (generally tuberculous) there is much effusion and thickening of the sheath, and formation of "melon-seed bodies" within the cavity.

**Symptomatology.**—Teno-synovitis due to

*over-use* most often occurs at the wrist. Slight pain and swelling develop, and on movement a very characteristic crepitus can be felt. The skin is not reddened. *Gonococcal* teno-synovitis causes much greater pain. It most commonly attacks the sheaths around the ankle-joint or—usually in women—at the wrist. The inflammation nearly always involves a group of sheaths and extends to the surrounding tissues, causing a diffuse oedematous swelling very tender to the touch and often with reddened skin over it. *Tuberculous* teno-synovitis produces an almost painless chronic swelling. Fluctuation can be elicited, and melon-seed bodies may sometimes be felt. The best example is compound palmar ganglion caused by tuberculous infection of the flexor sheaths in the palm. *Septic* teno-synovitis is common in the hand, and follows penetrating wounds, or may be secondary to a whitlow. The infection may rapidly spread to the palm and forearm. All the classical signs of septic inflammation are present, but the dense palmar fascia may cause the dorsum to be more swollen than the palm.

**Diagnosis.**—If the history as to recent strain, gonococcal infection, penetrating wound, or whitlow be ascertained, and the position and character of the swelling determined, no difficulty should be experienced in diagnosis.

**Treatment** for each variety may be summarized thus:—

*Sprain or over-use.*—Rest the part for a few days and apply pressure by wool and bandage or by strapping. Later, massage gently. In the early stages an evaporating lotion may assist.

*Gonococcal.*—Splint and foment during the acute stage, and treat the gonorrhoea. A course of vaccine treatment, using a stock or, preferably, an autogenous gonococcal vaccine, should be given. If a vaccine is not available, passive hyperæmia may be tried. After the acute stage is over, adhesions must be prevented by graduated massage and passive movements.

*Septic* (see WHITLOW AND INFECTIONS OF THE HAND; CELLULITIS).

*Tuberculous.*—Rest the part and aspirate the fluid, or make an incision to evacuate the melon-seed bodies. The administration of tuberculin (B.E.) and general antituberculous measures are indicated.

ZACHARY COPE.

**TERATOMA, CYSTIC** (see OVARIAN CYSTS).

## TESTAMENTARY CAPACITY

**TESTAMENTARY CAPACITY.**—This is measured by the presence or absence of mental impairment. It occasionally happens that an emergency will has to be drawn up by a doctor. This should be avoided whenever possible and the services of a lawyer requisitioned, but in exceptional cases the medical attendant may be forced by stress of circumstances to undertake the duty.

The evidence of the medical practitioner is particularly valuable in any inquiry concerning the mental capacity of a testator, and for this reason he should avoid witnessing a will in any case in which the testamentary capacity of the testator is likely to be called in question.

A testator must, at the time of making his will, understand the nature of the business upon which he is engaged, appreciate the possessions of which he is disposing, be able to call to mind the persons who have a claim upon his bounty, and understand the manner in which it is to be distributed.

The question of the sanity of a testator is one of fact, and there is no presumption that he is sane. If he is subject to delusions with regard to persons who would be the natural objects of his testamentary bounty, his will made under the influence of such delusions is invalid. But if a testator is subject to delusions which leave the general power of understanding unaffected and are wholly unconnected with his testamentary dispositions, such delusions do not affect his capacity to make a will. The fact of a testator being in an asylum is no bar to his making a perfectly sound will; the reasonableness of the will is the factor upon which its validity would be decided. A will made by a testator after he has been insane must be shown to have been made after his recovery or during a lucid interval. A lunatic, so found by inquisition, may make a will during a lucid interval, and the rational character of the will, if it is prepared by the testator without assistance, is evidence that it was made in a lucid interval.

Wills are often disputed on the ground that the testator was insane when the will was made, or that undue influence was exercised in getting the will framed in a particular way, and it is in cases of this class that the evidence of the doctor in attendance is particularly valuable. In one in which I was called upon to give evidence the degree of mental impairment was such that the loss of control over the bladder and bowels caused the testator no distress, and the loss of appreciation of

environment was such that he proposed, although in London, to go for a short drive to Bournemouth and back in a hansom cab. Yet, as the provisions were not unreasonable, the will was held to be good.

The only means available of estimating sanity or the amount of mental impairment is that of taking stock of a person's actions and of his conversation. These may or may not supply evidence of such an impairment of mind that the ability to direct a proper disposal of possessions is affected.

A *suicide* is not incapable of making a will. No person with acute mania or with an intense melancholia could be induced to sign a will.

No will of which the testator does not know and approve the contents can be valid. That a will is prepared by or on the instructions of the person taking a benefit under it is a circumstance raising much suspicion.

The influence of a person standing in a fiduciary relation to the testator may lawfully be exercised to obtain a will or legacy so long as the testator thoroughly understands what he is doing and is a free agent, and the burden of proof of undue influence lies upon those who assert it.

*To establish a case of undue influence* it must be shown that fraud or coercion has been practised on the testator in relation to the will itself, and not merely in relation to other matters or transactions. Undue influence is more easily established when there is evidence that the person influenced was of feeble mental capacity or in a weak state of health.

In forming an opinion as to the presence or absence of a "sound and disposing mind" a medical witness will consider the mental impairment which can be caused by any of the following conditions:—

1. The different degrees of congenital amentia.
2. Senile dementia.
3. Loss of will power and general mental decrepitude caused by illness.
4. Mental impairment due to mechanical injury to the brain, or to cerebral hæmorrhage, or to gross disease of the brain.
5. Insanity.

The *mental capacity of an aphasic person* requires consideration. It will be recalled that there are varieties of aphasia. Bateman, in "Aphasia and the Localization of the Faculty of Speech," p. 302, states that there is "no code or law as to the legal capacity of aphasics

## TESTAMENTARY CAPACITY

in this (Great Britain) or any other country, and each particular case would have to be considered on its own merits." Particular attention must be paid to the loss of general intelligence. There are cases on record in which the loss has been limited absolutely and entirely to the faculty of speech. In such a case it would not be impossible to divine the wishes of a testator.

In considering the testamentary capacity of an aphasiac, the attention of a court would be directed to the power of the testator to understand the nature of the property to be devised and the claims of the various persons upon his bounty, and to the question whether the will accurately represented his wishes. Usually it is very difficult for the court to satisfy itself that the claim of every person was present to the mind of the testator.

*Inebriety* has a bearing upon testamentary capacity, inasmuch as the will of an inebriate may afford evidence of the existence of delusions or of undue influence. A will executed by an intoxicated testator or by one suffering from delusions engendered by delirium tremens or from alcoholic dementia may on the face of it furnish evidence of loss of testamentary capacity. On the other hand, an inebriate in any of these states of mind may execute a perfectly good will, unless the degree of intoxication is such as to render him unable to comprehend the nature of his acts, or it is shown that insane delusions influenced him in the making of the will.

With regard to undue influence, the American Judge Earl states that "to make influence exercised over a testator undue and illegal it must be such as to destroy free agency; it is immaterial how little the influence may be if free agency is thereby destroyed."

Undue influence may be shown by all the facts and circumstances surrounding the testator, the nature of the will, his family relations, the condition of his health and mind, his dependence upon or subjection to the control of the person supposed to have wielded the influence, the latter's opportunity and disposition to wield it, and his acts and declarations. The fact that an inebriate, for example, has disposed of his property in an extraordinary manner is not sufficient to prove undue influence or to invalidate the will. It must be proved that the influence was actually exercised, and that the testator's mind was so impaired that the influence prevented the free expression of his will.

## TESTIS, NEW GROWTHS OF

For the principles of law and judicial pronouncements set out in this article I am indebted to "Theobald on Wills," and readers interested in case law are referred to that work.

F. S. TOOGOOD.

### TESTIS, INFLAMMATION OF (see ORCHITIS; EPIDIDYMITIS).

**TESTIS, NEW GROWTHS OF.**—With the exception of cysts of the epididymis, innocent tumours of the testis are extremely rare. The malignant tumours are carcinoma, sarcoma, endothelioma, and embryoma. Embryomata vary greatly in malignancy and in structure. They may be composed of tissues derived both from epithelium and from mesoblast, and thus are often known as "mixed tumours."

Sarcoma, either round or spindle-celled, may commence at any age. Carcinoma, usually of the soft or encephaloid variety, rarely scirrhous, generally occurs between the ages of 30 and 45. An imperfectly descended testicle is generally regarded as rather more prone to malignant disease than the normally placed organ.

**Symptomatology.**—The features of malignant testicular disease are rapid and progressive enlargement of the testis, usually painless, and with early loss of testicular sensation. Later there is generally some aching and dragging pain. At first the swelling is smooth and uniform, but later, when the tunica albuginea has been perforated, there are irregular bosses and projections. As a rule, the tumour is hard and firm, but it may be so soft as to suggest fluid. A small hydrocele is often present, and a history of injury may be obtained. The cord is thickened, at first owing to congestion, but later there may be nodules of growth. The skin of the scrotum, though stretched, is movable over the tumour until late, when it may be invaded and a fungous growth protrude. The prevertebral and lumbar glands are involved early, and may give rise to a palpable swelling in the upper part of the abdomen. The inguinal glands are not affected unless the skin of the scrotum is implicated. As the result of the glandular swelling there are likely to be, in the later stages, œdema of the legs owing to pressure on the inferior vena cava, and severe pain due to extension to the vertebræ and pressure on the spinal nerves.

**Diagnosis.**—The various forms of malignant disease cannot, as a rule, be distinguished



## TESTIS, SYPHILIS OF

from one another clinically. The tumour has often to be diagnosed from *syphilitic orchitis*, when a Wassermann reaction is of the greatest service; from a *hydrocele*, which may closely resemble malignant disease; and also, occasionally, from *hydrocele* and *tuberculous disease* without obvious caseation.

**Prognosis and treatment.**—Owing to the early glandular infection and the deep-seated position of the glands, the prognosis is very bad. The only treatment is free and early removal of the testis, with as much of the cord as possible, in the hope that the glands are not yet affected. Attempts have been made to remove the lymphatic glands and tract, but without much success.

PHILIP TURNER.

**TESTIS, SYPHILIS OF.**—Syphilis attacks the body of the testicle; the epididymis is rarely involved, though an epididymitis may occur in the secondary stage.

Syphilitic orchitis is a tertiary lesion, generally appearing from two to four years after infection, and may occur in congenital syphilis, usually between the ages of 6 and 12. In both the acquired and congenital disease there may be either a diffuse interstitial inflammation, or gummata, or a combination of the two. In the first, the tunica albuginea with the fibrous septa and the intertubular connective tissue are infiltrated with small cells which are subsequently converted into fibrous tissue; this contracts and leads to atrophy. Or the inflammation may be localized, giving rise to one or more gummata varying in size from a pea to a walnut, yellow in colour, and showing necrotic changes in the centre. These may become adherent to the skin, break down, and form gummatous ulcers which may lead to a "fungus testis." There is at first some effusion into the tunica vaginalis, but adhesions form later, with partial or complete obliteration of the cavity.

Syphilitic disease may terminate in resolution, or atrophy may result, the testicle becoming a small, bean-like, fibroid nodule; if bilateral, sterility and impotence are probable.

**Symptoms.**—Typically, there is slow progressive enlargement of one testicle, without pain and with loss of testicular sensation; rarely the onset is more acute. The epididymis becomes stretched out over the testis, which forms a firm, heavy, smooth, uniform, painless swelling that may attain the size of a fist or larger. When gummata are present the swelling is less uniform. The cord is not

## TESTIS, TORSION OF

affected. Both testicles are often attacked, but not as a rule simultaneously.

**Diagnosis.**—The diagnosis from *tuberculous disease* is generally easy, since the testis is affected in syphilis and the epididymis in tubercle. There may be a close resemblance to *malignant disease*, but the history and the result of a Wassermann reaction will generally settle the diagnosis.

**Treatment.**—The treatment is essentially antisymphilitic (*see SYPHILIS*) and, if it is carried out efficiently and without delay, the prognosis is good. Injections of salvarsan or some similar preparation should be given. Potassium iodide in increasing doses, especially when combined with mercury, is usually very effective. The treatment must be thorough and long-continued on account of the liability to relapse. The patient should be kept at rest in the early stages, and the testicle supported by a suspensory bandage. If the testicle is disorganized, or if hernia testis is present, it should be removed.

PHILIP TURNER.

**TESTIS, TORSION OF.**—Torsion of the testicle, or, rather, of the spermatic cord, is a rare condition, usually a complication of imperfectly descended testicle, and generally occurring before the age of 21. The testicle is probably always provided with a mesorchium, so that it is suspended from above and occupies a horizontal rather than a vertical position. Occasionally the twisted testicle is normally placed, but even then there is probably a mesorchium. The extent of the twist varies from half a turn to four turns. The testicle and the epididymis become engorged with blood, and this is soon followed by gangrene and infection. Bloodstained fluid is exuded into the tunica vaginalis.

**Symptoms and diagnosis.**—The twist may be brought about as the result of an injury, or it may be spontaneous. There is sudden severe pain, accompanied by vomiting and symptoms of collapse. Examination shows a tense, tender lump usually just below the external abdominal ring, the overlying skin being red and cedematous. The condition may closely resemble a *strangulated hernia*, but the abdomen is not distended, the vomiting is not faeculent, there is often pyrexia, and the testicle cannot be separated from the swelling. It may also have to be diagnosed from *acute epididymo-orchitis*.

**Treatment** is essentially operative. A free incision is made over the swelling and the

## TESTIS, TUBERCULOSIS OF

tunica vaginalis is opened. In a few early cases it may be possible to untwist the cord and fix the testicle in position by sutures; generally, however, it is advisable, even in early cases, to remove the testicle with the tunica vaginalis. If there is a communication between the tunica vaginalis and the peritoneum the sac should be traced up to the internal ring and there be ligatured before removal.

PHILIP TURNER.

**TESTIS, TUBERCULOSIS OF.**—Tuberculous disease nearly always commences in the epididymis, extending later to the body of the testis. Though generally occurring between the ages of 20 and 30 years, it may develop in children and also in older men. There is often a tuberculous history, and either old or active tuberculous lesions may be found elsewhere.

Infection generally takes place through the blood-stream, or may directly extend along the vas from the prostate. Indeed, tuberculous disease, when primary in the epididymis, tends to spread to the vas, vesiculæ seminales, and prostate, and conversely it may spread from these structures to the testicle. Hence tuberculous testicle must be regarded as part of a "genital tuberculosis," or, if the disease spreads to or from the urinary organs, of a "genito-urinary tuberculosis." Either the globus major or the globus minor may be first attacked—more commonly the former.

**Symptomatology.**—Typically, the onset is insidious. Pain is slight or absent, but enlargement may be accidentally discovered by the patient. Examination reveals one or more well-defined, slightly tender nodules either in the head or the tail of the epididymis while the body of the testis appears normal. Later, the whole epididymis becomes enlarged and has a typical craggy, nodular feel; there is probably some aching pain, and possibly a small hydrocele. Later still, increased swelling, with redness, œdema, and adhesion to the skin, indicates the formation of an abscess which, if untreated, bursts, forming one or more sinuses. These may heal spontaneously, or after treatment, but are always likely to reopen. A rectal examination will show whether the prostate and vesiculæ seminales are involved, and evidence of bladder and renal tuberculosis should be sought for. Sometimes the onset is acute and closely resembles gonorrhœal epididymo-orchitis.

**Diagnosis and prognosis.**—The diagnosis may be made by bacteriological examination

## TETANUS

of any urethral discharge and early abscess-formation. Except in the cases resembling gonorrhœal epididymo-orchitis, diagnosis is rarely difficult, though when the testicle is chiefly affected and caseation is absent there may be resemblance to a malignant growth. The disease may remain local for a long time, and the prognosis as regards life is less serious than in tuberculosis of many other organs. The chief dangers are dissemination and extension of the disease.

**Treatment.**—General treatment—cod-liver oil, iron, iodides—with residence in a suitable climate is of the greatest importance in all cases. Bier's treatment and injections of tuberculin have been successful in early cases. Abscesses and sinuses may be opened, scraped, and treated with iodoform emulsion. If sinuses persist, or if the disease is extensive but unilateral, the testicle should be removed with as much of the cord as possible; coexistent tuberculous disease elsewhere, if not extensive, is no contraindication. In those not infrequent cases where the disease is bilateral, or where after the removal of one testicle the disease appears in the other, the operation of epididymectomy may be performed.

PHILIP TURNER.

## TEST-MEALS (see GASTRIC CONTENTS EXAMINATION OF).

**TETANUS.**—A disease due to the action on the central nervous system of the toxins of the tetanus bacillus (Plate 5, Fig. 3, Vol. 1, facing p. 148). The bacilli, owing to the resisting power of the spores, are very hard to destroy. They may gain access to the body by even the smallest abrasion. They are commonly found in the intestines of normal horses, hence wounds soiled with road-surface or with fertilized earth are those most likely to be followed by tetanus. The bacilli have been found occasionally in gelatin and in catgut used in medical practice. The tissues of the body appear to offer a high resistance to the general dissemination of the tetanus toxin, for infection is much more likely to follow inoculation if other (e.g. pyogenic) micro-organisms, or foreign bodies, are also present in the wound.

Ten days is perhaps the most frequent period of incubation, but it varies from thirty hours to as many days, and in cases where prophylactic inoculation has been practised it has extended to a whole year. Premonitory symptoms are sleeplessness, restlessness, sweating,

## TETANUS

salivation, and muscular twitchings. Their recognition is important because their occurrence after a wound is an imperative indication for the administration of antitoxin. Experience gained in the late War showed beyond question that the value of prophylactic inoculation is immense, and that the earlier it is practised the better are the results to be expected. The dose should be at least 500 units, given subcutaneously or intravenously. If the wound be large and very dirty, 1,000 units may be given, and the dose should be repeated at weekly intervals until the wound begins to heal (*see* IMMUNITY). Excision of the wound and amputation of the injured member (both of which have been done) have been discarded on clinical grounds as useless procedures, and experimental evidence at least confirms their inutilty.

**Symptomatology.**—The muscular spasm which gives the disease its name usually begins in the masseters (trismus). The patient can no longer open his mouth—hence the popular name of the disease, *lockjaw*. The neck next becomes rigid, and then the facial muscles, giving rise to a peculiar facial expression (*risus sardonicus*). Next the trunk (*opisthotonos*) and the muscles of the limbs are attacked. The forearms and hands are little affected. Deglutition and respiration in severe cases may become impossible. The tetanic spasms are very painful. Exacerbations of spasm are excited by noise, light, handling, or attempts to swallow, and add greatly to the patient's misery. Sleeplessness is common and intractable, and a frequent pulse is of grave omen. Hyperpyrexia is an unusual but ominous sign. In cases ending favourably the spasm passes off in an order inverse to that of onset, *risus sardonicus* and trismus disappearing in six to eight weeks. Death is due directly to asphyxia, cardiac failure, and exhaustion.

It happens sometimes, especially in persons who have been prophylactically inoculated, that the manifestations are confined to the injured limb. This is called *local tetanus*. Infected wounds about the face may lead to an ipsilateral trismus and facial palsy (*cephalic or head tetanus*), or the spasm may involve muscles of the pharynx. The ensuing dysphagia presents a superficial resemblance to that of rabies (*tetanus hydrophobicus*). These modifications are not essentially different, and should be treated similarly. They are, however, somewhat less fatal.

**Prognosis.**—Pre-war statistics show a mor-

## TETANY

talitv in all cases of about 80 per cent. Nearly all deaths occur in the first fortnight, and patients surviving that period have a fair chance of ultimate recovery. The shorter the incubation period (fourteen days as critical), the worse the outlook.

**Treatment.**—Quietness is essential. Spasm may be relieved by morphia, urethane, and chloral (3 drachms in 24 hours per rectum). Antitoxin should be given, and given in big doses. The best method of administration is still under discussion. The intrathecal route is perhaps the best, and next the intravenous. Up to 24,000 U.S.A. units may be given intrathecally. A corresponding quantity of cerebrospinal fluid should be first withdrawn. The intrathecal dose should be given daily for four days, as the antitoxin rapidly disappears from the cerebro-spinal fluid. An intravenous dose of 15,000 c.c. should be given weekly.

Bacelli's treatment of daily injection with 2-per-cent. watery solution of carbolic acid (up to 5 gr. in one day) has proved disappointing. Meltzer and Auer recommended injections with magnesium sulphate (2 or 3 c.c. of a 25-per-cent. solution per kilo of body-weight); these too have been found useless. F. C. PURSER.

**TETANY.**—The principal objective symptoms of tetany are motor spasms of an easily recognized peculiarity. The conditions with which the spasms are associated are extremely various. The fundamental cause or causes are yet to be found.

**Symptomatology.**—Usually the spasms are bilateral, though one side may be the more affected. The muscles of the hands and forearms are involved most commonly, those of the face, feet, and other parts less frequently. The severity of the spasm varies; sometimes it can be overcome with little force, sometimes it is inflexible. The spasms are usually painful; attempts to overcome them add to the pain. They are intermittent, lasting for a few minutes, but may persist for hours or even for days. In severe cases they may never completely relax. The time separating attacks may likewise be measured in minutes, or hours, or days. The position assumed by the hand is characteristic. The thumb is opposed tightly to the index finger or pressed into the palm; the fingers are rigidly extended at the interphalangeal and flexed at the metacarpo-phalangeal joints—*accoucheur's hand*. The wrist and elbow are often flexed too. The foot assumes an equino-varus position, with the toes flexed

## TETANY

and the sole hollowed. In severe cases almost every muscle may be involved, even those of the larynx, pharynx, and eyes. There are certain peculiarities attached to these contractions. During a period of relaxation they can be brought on by pressure on the nerve-trunk or on the main vessel of the limb. This feature was described by Trousseau, and is known as *Trousseau's sign*. It is peculiar to, but not constantly present in, tetany.

The electrical excitability of the nerves is greatly heightened (*Erb's sign*), so that a contraction can be induced in a muscle by a far smaller current than normal. This sign is very constant. Not so constant, and not pathognomonic of tetany, for it occurs in other conditions, is *Chvostek's sign*—a heightened excitability of the nerve trunks to mechanical stimulation. It is most easily demonstrated by percussion of the facial nerve.

Pains and paræsthesiæ may precede the onset of an attack. The irritability of sensory nerves, like that of motor nerves, is often increased, and slight pressure on a nerve, which in a normal individual would be appreciated only locally, may be felt radiating unpleasantly throughout the area of distribution of the nerve (*Hoffmann's sign*).

General constitutional signs, as fever, emaciation, etc., are due only to concomitant conditions. These conditions are very numerous. A mild form of tetany (if tetany it may be called) occurs fairly often as carpo-pedal spasm in young children in whom one can find nothing wrong but a slight gastric or intestinal trouble. Laryngismus is often associated. Such cases are mild and get well rapidly on simple diet and an aperient, though there may be recurrence the next time the child's digestion is out of order. Tetany occurs in rickets; a lack of calcium salts in the body is one of the causes to which it has been attributed. It occurs also before, during, and after infectious diseases—typhoid, influenza, measles, and pneumonia—and in nephritis. It may accompany pregnancy and lactation. It has followed the administration of phosphorus, male-fern, chloroform, spermin, and other drugs. These loudly suggest a toxic origin. It has occurred in epidemic form in Vienna and other large towns, especially in the spring. Endemic tetany affects cobblers and tailors especially, and at one time the condition was regarded as an occupation neurosis—like writer's cramp. It has followed removal of the thyroid gland; but it is probable that in these cases it was due to the removal of

## THROMBOTIC PHLEBO-ARTERITIS

the parathyroids rather than of the thyroid. The parathyroids are believed to have a toxin-neutralizing effect, their ablation leaving the subject defenceless against unnameable toxins. Parathyroid extracts, however, have proved of little value in treating tetany. A particularly severe form of tetany, *gastro-tetany*, occurs in cases of gastrectasis, especially after lavage has been carried out. But here again no definite toxin has been isolated. In some of the reported cases there has been an unusually high number of cells, red and white (the latter preponderatingly polynuclear), in a blood-film. Recent observations have shown that the excretion of guanidin is much increased in tetany, and indicate that the symptoms may be due to the effect of this substance on the tissues.

**Treatment.**—Bromides, chloral, and morphia will help to allay the severity of the spasms. But the underlying cause must be sought, and treatment directed to its removal. The prolonged administration of calcium has frequently a favourable influence on the disease.

F. C. PURSER.

**THALAMIC SYNDROME** (see PAIN, CENTRAL).

**THIRD NERVE, PARALYSIS OF** (see OPHTHALMOPLÉGIA).

**THOMSEN'S DISEASE** (see MYOTONIA CONGENITA).

**THREADWORM** (see INTESTINAL WORMS).

## THROMBOTIC PHLEBO-ARTERITIS.

—An inflammatory thickening of the walls of arteries or veins, causing a diminution of the lumen, which may proceed to complete obliteration.

**Etiology.**—The disease is rare, more common in males, and comparatively frequent in male Polish and Russian Jews. Apparently it occurs chiefly between the ages of 20 and 40. Syphilis is perhaps the most certain etiological factor, but tobacco is thought by some to have an important influence, many cases having been recorded amongst cigarette-makers. It is sometimes associated with vascular hypoplasia.

**Pathology.**—The thickening of the arterial walls is due to cellular proliferation of the endarterium and to hypertrophy of the media and adventitia. The obliteration of the lumen seems to be due to organization of a thrombus. The lesion, often symmetrical, occurs especially

## THYROGLOSSAL DUCT, AFFECTIONS OF

in the lower limbs, less commonly in the upper, and begins at the extremity of the limb. The visceral and other arteries may also be involved.

**Symptoms.**—The premonitory symptoms are indefinite pains in the limbs, intermittent cyanosis, cramps, and especially coldness and numbness. These symptoms tend to become permanent. In some cases typical symptoms of intermittent claudication occur (*see ARTERIAL DEGENERATION*). After months or years trophic disturbances appear. According to Bürger, in the pendent position a bright-red blush of the anterior part of the foot comes on, simulating erythromelalgia, a blister or ulcer develops on a toe, accompanied at times by a discoloration in its neighbourhood, and finally gangrene ensues.

**Diagnosis.**—This depends on the super-vention of gangrene, generally symmetrical and ascending, of the extremities, without very obvious cause, in young subjects.

**Treatment.**—In the case of a patient who has been treated for gangrene arising from this condition, rest of the affected limb is important. Careful massage is useful. Influences, such as cold, which exert a vaso-constricting effect should be avoided. (For treatment of the gangrene, *see GANGRENE OF EXTREMITIES*.)

OLIVER K. WILLIAMSON.

**THROTTLING** (*see ASPHYXIA*).

**THRUSH** (*see STOMATITIS AND GLOSSITIS*).

**THYMIC ASTHMA** (*see LYMPHATISM*).

**THYMUS GLAND, ENLARGEMENT OF** (*see LYMPHATISM*).

**THYROGLOSSAL DUCT, AFFECTIONS OF.**—This embryonic canal commences at the foramen cæcum, runs down the centre line, passing behind the body of the hyoid bone, in front of the thyrohyoid membrane and the thyroid cartilage, to terminate in the isthmus of the thyroid gland, which is developed from its lower end. The whole of this tract is obliterated in the course of development, but rarely portions of it may be seen giving rise to cyst-formation, to development of accessory thyroids or dermoids, or to the formation of a fistula.

**Cyst-formation.**—The usual position in which the cyst is found is either at the base of the tongue or just in front of the thyrohyoid membrane, when it commonly extends upwards behind the body of the hyoid bone. It usually

develops from the first to the sixteenth year. Dermoids may occur anywhere along the course of the duct.

**Accessory thyroids.**—These may be found in any part of the tract, but are most likely to occur at the base of the tongue, varying in size from  $\frac{1}{2}$  in. to 2 in. in diameter.

**Dermoids** are usually tense, round, fluctuant swellings in the middle line at the base of the tongue, where they generally become pedunculated.

**Fistula** is nearly always situated just above the thyroid isthmus, unless it results from the incomplete removal of a thyroglossal cyst, when it is usually found in front of the thyrohyoid membrane. The discharge may be slight or it may be copious, leading to irritation and inflammation round the orifice.

**Prognosis and symptoms.**—These conditions are all harmless, and are better left alone. Cysts give rise to no symptoms, but for cosmetic reasons many patients demand a removal. The smaller thyroids at the base of the tongue are innocuous, but the larger may give rise to obstruction both to respiration and deglutition. Fistulæ, as a rule, do not give much trouble unless, as stated above, the discharge is copious, when there may be considerable irritation and inflammation round the opening.

**Diagnosis.**—There is no difficulty in the case of fistulæ. The dermoids at the base of the tongue are usually pedunculated, whereas the accessory thyroids are not; they also differ from the latter in being fluctuant. In regard to cysts, the chief difficulty lies in the diagnosis between them and a subhyoid bursa. The chief points are that a bursa is more central, its sac is more strictly limited, and it is more prominent below the hyoid.

**Treatment.**—Where possible, do nothing. If, however, the fistula is giving trouble it must be dissected out in its whole length, the body of the hyoid being, if necessary, divided to remove the part behind it—the most troublesome part of this operation. Injections of iodine, etc., are useless. In the case of a dermoid or accessory thyroid at the base of the tongue, the former can usually be removed with a snare. The same can often be done for a thyroid, or, if this is not feasible, it can be removed by a transhyoid pharyngotomy. If a cyst is removed, it must be removed in its entirety, as the leaving behind of any part of its wall will invariably lead to a fistula; but operation should, if possible, be avoided.

J. GAY FRENCH

## TICS AND HABIT-SPASMS

**THYROID GLAND, AFFECTIONS OF**  
(see EXOPHTHALMIC GOITRE; GOITRE; CRETINISM; MYXŒDEMA; ORGANO-THERAPY).

**TIG-DOULOUREUX** (see NEURALGIA).

**TICK FEVER** (see RELAPSING FEVER).

**TICS AND HABIT-SPASMS.**—According to the best authorities, a tic may be defined as “a co-ordinated purposive act, provoked in the first instance by some external cause or by an idea; repetition leads to its becoming habitual, and finally to its involuntary reproduction without cause and for no purpose, at the same time as its form, intensity, and frequency are exaggerated: it thus assumes the characters of a convulsive movement, inopportune and excessive; its execution is often preceded by an irresistible impulse, its suppression associated with malaise. The effect of distraction or of volitional effort is to diminish its activity; in sleep it disappears. It occurs in predisposed individuals, who usually show other indications of mental instability” (Meige and Feindel).

If this definition is followed closely—which is the more desirable as tic and analogous terms for involuntary movements are often used in a loose and unscientific fashion—it is at once seen that a tic, or habit-spasm, is essentially a psycho-motor disturbance; it is the caricature of a normal purposive movement, of cortical origin. The expression “habit-spasm” should be relinquished, for the term “spasm” is now, after Brissaud, to be clearly distinguished from tic. A spasm is the motor reaction consequent on stimulation of some point on a reflex spinal or bulbo-pontine arc. The irritation provocative of the spasm is itself of pathological origin, and no spasm can occur without it. A simple illustration will make the difference clear. An irritant particle on the conjunctiva will cause blinking; the blinking movement is a spasm of the muscles concerned. Should the blinking movement continue though the source of irritation has vanished it becomes habitual, and should it eventually occur in season and out of season, inopportune and excessive, it becomes a tic. Spasms may or may not be painful; tics are painless. The involuntary blinking of the eyelids owing to irritation is a purely reflex phenomenon in which the cortex has played no part, whereas in tic it is the activity of the cortex which first makes the movement habitual.

A tic, further, is more than a mere trick or mannerism, a so-called stereotypy or stereotyped act. The essence of a stereotypy is that it occurs only at certain times, that the patient can control it if he cares to, that it is not exaggerated in form, frequency, or intensity. Observe the player who takes a hard ball at tennis, or the pianist who performs a difficult passage, or the cueist who makes a delicate shot: the effort is accompanied by a facial grimace, a biting of the lip, a frown, always repeated in the same way, only at such a time, and never exaggerated in degree. These are tricks or mannerisms, but not tics.

**Etiology.**—Charcot said that “not all who would may tic,” thereby indicating that mere repetition of a movement will not make the subject a *tiqueur*. The subjects of tic are found without exception to be persons with a neuropathic heredity, who show other signs of mental instability or inequality; they are psychically predisposed. Tics occur in both sexes and at any age, except in very young children. In the predisposed, imitation, environment, anxiety, worry, fatigue, mental or physical strain, are all etiological factors that may be of significance.

**Symptomatology.**—The motor reaction constituting a tic may be slow or quick, deliberate or abrupt. The tic movement may be a mild contraction or a violent convulsion. Tics may be single or multiple. A “tonic” or “attitude” tic may undoubtedly be met with. *Ex hypothesi*, there may be as many tics as there are movements of a purposive nature; any of the latter may form the basis of a tic. Hence any exhaustive description of their symptomatological varieties is out of the question.

We meet with facial tics of all sorts: tics of winking, blinking, sniffing, grunting, licking, sucking, biting, pouting, grimacing, nibbling, mumbling, etc. Tics of the neck and head are very common—head-nodding and head-tossing tics, tics of affirmation and of negation, tics of salutation; every sort of head movement may occur as a tic. Some forms of wry-neck are pure tics.

Tics of the shoulders, trunk, arms, hands, fingers, may occur—scratching, rubbing, scraping, stroking, tapping, etc. All varieties of inspiratory and expiratory sounds may constitute tics—whistling, coughing, sobbing, hic-coughing, and so on. Some tics are much more complex—co-ordinated tics, as they are called. Thus, tics of speech may be found,

## TICS AND HABIT-SPASMS

in which the patient ejaculates some syllables or words in a meaningless but irresistible way, interlarding his ordinary conversation; if the expressions are obscene or blasphemous, the tic is known as coprolalia. Some trunk and body tics are very complex, e.g. tics of genuflexion.

Mention may also be made of the variety known as the "disease of convulsive tics," or Gilles de la Tourette's disease. The tics are multiple and violent; they are commonly accompanied by echolalia and coprolalia; the patient's mental condition is unmistakably that of the degenerate, and the prognosis is unsatisfactory and often grave, as insanity not infrequently develops.

Tics, apart from their occurrence monosymptomatically, are sometimes met with in the course of other diseases. Tic is closely related to psychasthenia; it is also related to epilepsy. In defectives of all sorts tics are common, and in idiots and demented more particularly.

The **diagnosis** of tic has already been touched upon in the paragraphs on its definition where allusion is made both to spasm and stereotyped acts. The other chief morbid condition from which tic may have to be differentiated is ordinary *chorea*. Sometimes the diagnosis from *chorea* is difficult, especially in cases of unilateral *chorea* which are becoming chronic. In most instances, however, the presence of concomitant symptoms in *chorea* enables the observer to distinguish the condition. Moreover, the actual type of motor reaction in *chorea* is amorphous, purposeless, inco-ordinate; tic is subjectively purposeful and repetitive.

**Prognosis** depends to a large extent on the patient's mental state. The greater his mental imperfection the more likely are his tics to be chronic. A cure is certainly possible in some cases, but, speaking generally, while improvement under treatment is frequent, relapses are equally frequent.

**Treatment.**—The general management of the case is of much importance. Not infrequently the environment of home, especially if the parents are themselves neuropaths, is distinctly unfavourable. It may be desirable, therefore, to send the patient to other surroundings for a time. In many cases, further, it is important to give him some aim in life, to start him on work that will absorb his attention, to develop in him a zest for some hobby that will occupy his spare hours. Simple hygienic measures calculated to improve the

general "tone" may be necessary. Examination of a tic patient should always include a careful routine examination of eyes, ears, teeth, nose, etc., for the detection of any abnormality that may have acted as a focus of irritation.

Treatment by means of sedative drugs sometimes proves of value, e.g. small doses of bromide given over a long period. In many other cases, drug treatment is useless. Local massage and electrical treatment and Weir-Mitchell methods are practically of no avail.

Undoubtedly re-education is the best method of treatment to adopt. It consists mainly in combining an endeavour to obtain immobility on the part of the patient with the systematic employment of voluntary exercises, to include the muscles usually involved in the tic or tics of which he complains. The patient sits or stands in front of a mirror and endeavours to maintain absolute immobility, it may be for only a second or two. He increases, if he can, the duration of this immobility by a few seconds at each séance, carried out several times daily, and under the encouragement of the physician he frequently succeeds beyond all expectation by this simple means. He also carries out systematic graded exercises; he is made to innervate the affected muscle-groups individually, so many times at each séance, and when his tic interrupts he starts afresh. By following a detailed schedule and under the constant supervision of the physician or other responsible person he may attain extremely good results.

Apart from individual exercises for particular muscles, the patient's will may be re-educated by general respiratory exercises and by calisthenic and gymnastic methods. Still another form of exercise consists in "mirror drill," which has nothing to do with actual mirrors, but means simply the bilateral use of symmetrical muscles in the performance of set exercises. In all cases of tic confined more or less to one side it is an excellent plan to apply muscular exercises to both sides of the body simultaneously; the regularity with which they are performed on the sound side will have a corrective influence on the abnormal movements of the affected side.

S. A. KINNIE WILSON.

**TINEA** (see RINGWORM).

**TINEA FLAVA** (see TINEA (PITYRIASIS) VERSICOLORE).

**TINEA MARGINATA** (see RINGWORM).

## TINEA (PITYRIASIS) VERSICOLOR

**TINEA (PITYRIASIS) VERSICOLOR.**—A superficial infection of the epidermis by a mould fungus (*Microsporon furfur*), characterized by the formation of brown scaly macules, which enlarge and coalesce to form extensive patches on the trunk. (PLATE 28, Fig. 6, facing p. 138.) *Tinea flava* (*Malassezia tropica*) is a similar condition found in the tropics.

**Etiology and pathology.**—The disease occurs chiefly in male adults, and since free sweating is a predisposing factor, it is often seen in patients suffering from phthisis. Probably thick flannel clothing and abstention from frequent ablutions also conduce to its development and perpetuation. It has a very low degree of infectivity, and rarely occurs in more than one member of a family. The fungus is easily demonstrated by lightly scraping the skin and examining the scales under a one-sixth objective in liquor potassæ. When the preparation has cleared, an abundance of mycelium consisting of short strands with rounded ends, intermingled with round spores which have a tendency to become massed together like bunches of grapes, is seen. The fungus is very difficult to cultivate, but a few successes have been obtained by using epidermin-agar as a medium. There are only a few instances of successful experimental inoculation on record.

**Symptomatology.**—The disease begins with one or more brownish macules, often on the sternal region or on the abdomen or back. These macules gradually extend peripherally and coalesce with others, forming extensive irregular-shaped areas often involving large parts of the trunk. In exceptional cases the arms or thighs are invaded. The disease spreads very slowly, and, as itching is absent or extremely slight, patients often disregard it until after a duration of several years it becomes so extensive as to suggest the desirability of treatment.

**Diagnosis.**—The peculiar brownish-yellow colour—the slight branny desquamation, the ease with which the scales can be detached by the finger-nail or a sharp instrument, the distribution on covered parts of the body, and the large extent of surface often affected, are characteristic features, and serve to distinguish the disease from the true pigmentation of chloasma, Addison's or Graves's disease, arsenical pigmentation or that associated with leucoderma, syphilis, and other pigmentary conditions. If there is any doubt, microscopical examination will at once make the diagnosis clear.

**Prognosis and treatment.**—When proper

## TINNITUS AURIUM

attention is given to the treatment of tinea versicolor, there is no difficulty in effecting its removal. The tendency of the disease to relapse should be combated by thorough disinfection of the underclothing and by persisting in the treatment after apparent cure. The skin should be washed night and morning with soft soap and hot water, and an antiparasitic lotion applied immediately afterwards. Hyposulphite of soda, one or two drachms to an ounce of water, is a favourite application; sulphurous acid in similar strength is preferred by some. An ointment consisting of precip. sulphur 15 gr. and salicylic acid 10 gr. to an ounce of soft paraffin is also efficacious, and may be used to replace the sulphur lotion at night. No internal medication is required.

S. E. DORE.

**TINNITUS AURIUM.**—Tinnitus aurium is a subjective sensation of sound due to irritation of the cochlea. This irritation may, of course, be induced by almost any disease in the ear. It is also sometimes the result of disease elsewhere; this is the case in the blowing tinnitus of which anæmic girls frequently complain, and in the throbbing or pulsating noises that trouble people with arterio-sclerosis or indigestion.

The first duty when complaint is made of "noises in the ears" is to determine the cause—a procedure which not infrequently necessitates a complete overhauling of the patient.

Naturally, the organ to which attention should first be paid is the ear, and, as tinnitus may be a symptom of practically any of its diseases, the reader is referred to the article on EAR, EXAMINATION OF, for further information upon this point.

Affections of the ear having been excluded, we turn next to the patient's general condition, particular attention being paid to the state of the heart, blood-vessels, blood, and urine.

Frequently causes are encountered, however, in which no cause whatever can be discovered to account for the tinnitus and the only explanation we can offer is that it is of a "functional" or "nervous" character.

At the same time, it should be remembered that tinnitus is often the earliest symptom of a disease which, later on, will cause deafness. So that it is highly inadvisable to allow ourselves to be satisfied with the diagnosis of *tinnitus nervosa*.

Tinnitus is often a disturbing, sometimes a distressing, and occasionally a dangerous symptom *per se*, from which the patient desires to



## TOE-NAIL, INGROWN

be liberated. In such cases a correct diagnosis of the underlying disease too often advertises our incompetence to cure it, so that we are little better off than if no diagnosis at all were attempted.

The danger of tinnitus in certain cases lies in the fact that the indefinite subjective sounds or tones gradually assume the definite character of hallucinations, and end in insanity. In addition to that risk, most otologists of experience are able to recall cases of suicide as a result of the depression due to the incessant, persistent, and irremediable noise in the ears, or in the head.

Medicinally, the best internal remedy for tinnitus is a mixture of bromide and nuxvomica :—

R<sup>y</sup> Sod. brom. gr. x.  
Tr. nuc. vom. ℥vi.  
Aq. ad 3ss.  
T.d.s., or more often.

If this fail, other nerve sedatives may be tried. I have frequently found nitroglycerin (liq. trinitrini, 1 min.) to be beneficial.

The disease in the ear, if the ear be at fault, must, of course, receive its proper treatment.

When tinnitus becomes a terror, and is threatening the reason or the life of the unhappy sufferer, recourse may legitimately be had to an operation upon the labyrinth with the express object of destroying the end-organ of hearing in the cochlea. This operation has been performed with satisfactory results in such extreme cases.

DAN M'KENZIE.

## TOBACCO AMBLYOPIA (see AMBLYOPIA).

**TOE-NAIL, INGROWN.**—A painful, crippling paronychia which affects usually the big toe. It is caused by wearing boots too narrow for the foot, and is predisposed to by cutting the free extremity of the nail in a curve instead of straight across, as it should be cut. Generally the lateral edge of the nail is forced against the soft tissue of the toe, which suffers to the extent of ulcerating. The little sore lies close to the edge of the nail and is at the bottom of a cleft formed by the overlapping swollen skin and subcutaneous tissue. Sepsis supervenes and pus exudes from the wound groove. The condition lasts for long, as the drainage is poor. Very soon granulations from the floor of the ulcer grow up and protrude. The infection may extend to beneath the nail, giving rise to a painful subungual whitlow.

**Treatment.**—Rest with the foot kept raised

## TONGUE, ACUTE OEDEMA OF

on a chair is the first essential, and when boots are again worn they must be of sufficient width. Early cases can be cured by packing a thin wisp of cotton-wool sprinkled with boric-acid powder under the overhanging fold, and changing this daily. The more advanced cases are only cured by operation. Removal of the nail, recommended by some, usually

affords only temporary relief, as the condition is liable to recur. Subungual pus is the only indication for nail removal. Better by far is it to proceed as follows. If there is acute inflammation present, apply fomentations until the acute stage has subsided and the lesion is comparatively clean. Then under local or general anæsthesia remove a piece of the toe, including the hypertrophied fold and a part of the nail. In shape the excised piece resembles a segment of an orange (Fig. 95); and the nail matrix on the exposed terminal phalanx corresponding to the removed section of nail is also taken away. The soft parts are then sutured to the cut



Fig. 95.—Incision for ingrown toe-nail.

edge of the nail by two silkworm-gut sutures carried in a small curved Hagedorn needle. A narrow nail is left. The condition does not return.

If local anæsthesia be employed, novocain 1 per cent., without adrenalin, should be used. It is injected in the neighbourhood of the four digital nerves after a narrow piece of rubber tubing has been tightly adjusted around the base of the toe to act as a tourniquet. The operation is done fifteen minutes after the injection.

C. A. PANNETT.

**TONGUE, ACUTE OEDEMA OF.**—Acute parenchymatous glossitis may be due to stings of insects; to wounds, especially with a retained foreign body; or to infection extending from superficial ulcers, from the tonsils, or from septic teeth, especially during one of the infective fevers. It may also occur with Ludwig's angina. Sometimes there is no obvious cause.

**Pathology.**—Occasionally half the tongue only is involved. The usual termination is

## TONGUE, NEW GROWTHS OF

resolution, possibly with some fibrosis. Suppuration is unusual, and gangrene very rare.

**Symptoms.**—The tongue rapidly swells; it fills and protrudes from the mouth, is covered with mucus, and may show superficial excoriations. There is profuse salivation, the breath is foul, the cervical glands are enlarged, and the temperature is raised. The constitutional symptoms are severe. Pain, dysphagia, and dyspnoea are present, and unless the swelling is relieved asphyxia may prove fatal.

**Prognosis.**—Though recovery is usual, a fatal broncho-pneumonia may develop, or death may occur from septicæmia.

**Treatment.**—In the early stages a purge should be given, also ice to suck, and antiseptic mouth-washes. If there is obstruction to breathing, two free incisions should be made into the muscular tissue. Threatened asphyxia will call for tracheotomy, but, since this increases the danger of broncho-pneumonia, it should not be done unless absolutely necessary.

PHILIP TURNER.

### TONGUE, MINOR AFFECTIONS OF (see STOMATITIS AND GLOSSITIS).

**TONGUE, NEW GROWTHS OF.**—Both innocent and malignant new growths occur in the tongue. Of *innocent* tumours, many varieties are met with, but they are all rare and generally of slight importance. The chief are: *angioma*, either capillary or cavernous; *lymphangioma*, usually involving the anterior two-thirds and known as macroglossia; *papilloma*, which may resemble epithelioma; *lipoma*, single or multiple; and *cysts*, either superficial mucous retention cysts, or cystic tumours in the root of the tongue derived from the thyroglossal duct. The *malignant* growths are *sarcoma*, which is extremely rare, and *epithelioma*, which is very common.

#### EPITHELIOMA

**Etiology.** Epithelioma is much more frequent in men than in women, and generally occurs between the ages of 40 and 60. The usual site is the lateral margin in the anterior two-thirds, more rarely on the dorsum or the under-surface. In a large proportion of cases it is clearly the result of some long-continued irritation such as from the edge of a broken tooth or ill-fitting denture, while in others it starts in some "pre-cancerous" lesion, most frequently chronic superficial glossitis, but occasionally the scars of other tertiary syphilitic lesions, or in dental ulcers.

**Symptomatology.**—An epithelioma may begin (a) as an indurated nodule extending into the deeper tissues, (b) as an indurated fissure, (c) as a warty growth, or (d) an ulcer may be present from the first. In any case, ulceration with induration and rapid extension soon become the characteristic features. A typical epitheliomatous ulcer has raised and everted edges and an irregular sloughy base with surrounding induration extending deeply into the tissues. Unless efficiently treated the disease soon extends to the floor of the mouth, jaw, fauces, and tonsil. The submaxillary lymphatic glands and the deep cervical group, especially those at the bifurcation of the carotid, are involved at a very early stage, occasionally on both sides of the neck. Later they enlarge considerably and, partly owing to septic infection, become adherent to adjacent structures, including the skin, and break down, forming foul, sloughing, fungating ulcers. Secondary deposits in the viscera, though sometimes found, are distinctly unusual.

Even in the early stages there is severe pain, often shooting to the ear, while in the later stages the sufferings of the patient are intense. Salivation is profuse, and in advanced cases there is much foul, bloodstained discharge. Owing to the induration, mobility is at first limited, and in the late stages the tongue cannot be protruded, rendering both speech and swallowing difficult and painful. Towards the end there is great wasting and cachexia, with septic absorption from the ulcerating surfaces.

**Diagnosis.** Early diagnosis is of the greatest importance, but occasionally presents considerable difficulties. Syphilitic ulcers, both primary and tertiary, tuberculous and dental ulcers, and papillomata may all closely resemble epithelioma. In doubtful cases a portion of the ulcer should be excised for histological examination. Age may be misleading, for epithelioma may occur at the age of thirty, or even before.

**Prognosis.**—Prognosis is extremely bad, even when the disease is treated at an early stage. Recurrence in the tongue after operation is unusual, but the complete removal of the infected lymphatics is extremely difficult, and hence only too often the operation is followed by the appearance of deep-seated growths in the neck. Death is commonly due to chronic septic absorption and exhaustion, but occasionally to septic broncho-pneumonia or to secondary hæmorrhage from the lingual or one of the large arteries of the neck.

## TONGUE, PARALYSIS OF

**Treatment.**—The treatment is removal of the growth with the affected half of the tongue and of the lymphatic glands as soon as the disease is recognized. The tongue is best removed by Whitehead's method. It is split in the midline with scissors, and after the mucous membrane of the floor of the mouth has been divided and the lingual artery secured, the affected half is cut across well behind the growth. Any bleeding vessels are secured and the raw surface is diminished by a few catgut sutures. The site of the growth may render division of the jaw or cheek necessary for its satisfactory exposure. The glands are removed by a curved incision extending from the symphysis to just behind the angle of the jaw; a second incision extends from this along the sterno-mastoid. By turning back the skin a free exposure of the submaxillary and carotid triangles is obtained. The submaxillary salivary and lymphatic glands are removed, as well as the glands in front of the carotid sheath. The operation is a severe one. Intratracheal anaesthesia prevents respiratory obstruction during the operation and diminishes the liability to inhalation pneumonia. By some it is advised that the operation should be done in two stages at an interval of ten to fourteen days, but opinions differ as to which should be done first. When both tongue and glands are operated upon at the same sitting, the glands should be removed first, as the lingual artery can then be secured before the tongue is divided. The chief objection to the two-stage method is that the patient may refuse the second operation—hence the physical condition and determination of the patient have both to be taken into account.

When the disease of the tongue is extensive and complete removal doubtful, diathermy (q.v.) may be employed with benefit in some cases. If the disease is recurrent or inoperable, radium or X-rays may be tried, or injections of colloidal selenium, though usually with little effect. In these hopeless cases opium will be required to give the patient some relief.

PHILIP TURNER.

**TONGUE, PARALYSIS OF.**—All the muscles of the tongue are supplied by the hypoglossal nerves, and those of the same side are paralysed and atrophy when any lesion injures either the one nerve or its nucleus. The nucleus may be involved in poliomyelitis, syringomyelia and other local lesions of the bulb, and the

## TONGUE, SYPHILIS OF

nerve may be damaged by traumata, inflammatory foci, and tumours.

The symptoms of a *unilateral* atrophic palsy are slight; there may be a slight disturbance of articulation and some difficulty in collecting food from the cheek of the same side. While in the mouth the palsied side stands higher, and when protruded the tongue deviates and its tip is often curved towards the affected side. As atrophy supervenes, the mucous membrane that covers it becomes puckered and wrinkled, and fibrillation may be observed.

*Bilateral* atrophic palsy occurs chiefly as a part of chronic bulbar paralysis; articulation is impaired, mastication becomes difficult, as the tongue cannot keep the food between the jaws, and swallowing is affected, as it cannot bring the bolus promptly back to the pharynx. Sensation and taste are undisturbed.

Supranuclear lesions produce a spastic paresis without wasting and fibrillation; when unilateral it is generally part of a hemiplegia. Bilateral spastic weakness is found in pseudo-bulbar paralysis; it produces disturbances of articulation, mastication, and deglutition.

GORDON HOLMES.

**TONGUE, SYPHILIS OF.** — Primary, secondary, and tertiary lesions on the tongue may all occur in the acquired disease; the last two also in the congenital.

A **primary sore** is rare; it usually occurs in men, on the anterior part of the tongue. It begins as a papule, which increases in size and becomes indurated and ulcerated. There is considerable and painful enlargement of the lymphatic glands.

**Secondary lesions** are common. Mucous patches, small greyish areas of sodden and heaped-up epithelium, associated with similar lesions on the mucous membranes of the cheeks and lips, are frequently seen. Small papules and superficial but painful ulcers may also occur. Usually there are carious teeth with oral sepsis; secondary manifestations will also be found elsewhere.

The following **tertiary lesions** are often met with, either singly or combined: (1) *Chronic superficial glossitis*, an insidious and chronic inflammation of the mucous and submucous layers, as the result of which white patches, of irregular shape and varying extent, known as *leucoplakia*, and due to thickening of the epithelium, are produced. These may be thick, horny, and warty, and are then known as *ichthyosis linguæ*. Elsewhere the papillæ

## TONGUE, TUBERCULOSIS OF

and mucous membrane may gradually be destroyed, and replaced by thin scar tissue giving a characteristic red glazed appearance. (2) *Chronic parenchymatous glossitis* terminating in fibrosis and contraction, with the production of irregular ridges and fissures. This and the previous form often coexist. The tongue is painful, tender, and easily irritated even by food. (3) *Gummata* may be single or multiple. They appear as painless elastic swellings in the muscular tissue on the dorsum, and are liable to break down and form (4) *gummatous ulcers*, round or irregular in shape, deep, with a sloughy base and foul discharge. Induration is slight, and there is little or no pain.

**Diagnosis.**—Syphilitic lesions may be difficult to diagnose. With a primary sore the rapid enlargement of lymphatic glands is suggestive, and the histological examination of scrapings for the spirochete, or the appearance of secondaries, should be conclusive. Secondary lesions usually present no difficulty, but a tertiary lesion may closely resemble epithelioma or, occasionally, tuberculous ulceration. A Wassermann reaction and, in doubtful cases, histological examination of an excised portion will clear up the diagnosis.

**Prognosis.**—While efficient treatment may lead to the complete absorption of a gumma, it must be remembered that the scarring of tertiary lesions is permanent and that epithelioma so frequently develops in these that they are recognized as "pre-cancerous" conditions.

**Treatment.**—In all stages the treatment is essentially that of syphilis (q.v.). In addition, attention should be directed to overcoming oral sepsis by the extraction of carious teeth and the use of antiseptic mouth-washes. Owing to their liability to the development of epithelioma, tertiary lesions, if of suspicious appearance, may, if localized, be excised.

PHILIP TURNER.

**TONGUE, TUBERCULOSIS OF.**—An uncommon disease, usually occurring between the ages of 20 and 30, and practically always secondary to tuberculous disease of the lungs and larynx. Lupus of the tongue is also occasionally seen with lupus of the face or nose.

**Symptomatology.**—Lingual tuberculosis may begin as a tuberculous nodule which breaks down, or ulceration may be the first lesion. One or more ulcers may be present, and the usual site is on the dorsum near the

## TONGUE, ULCERATION OF

tip. It may also occur in the lymphoid tissue in front of the epiglottis, and occasionally on the lateral margin of the tongue. A typical tuberculous ulcer has an irregular outline with well-defined edges and a pale sloughy base with muco-purulent discharge. It is at first a superficial erosion, but later extends more deeply; there is little or no induration to be noted, and the movements of the tongue are not impaired. At the side of the tongue a tuberculous lesion may appear as a crack or fissure with hard edges, closely resembling an epitheliomatous or a dental ulcer.

The ulceration is extremely painful and, with the accompanying disease of the larynx, may render swallowing almost impossible. The patient is weak and wasted. The cervical glands are usually not enlarged.

**Diagnosis.**—Tuberculosis may have to be diagnosed from epitheliomatous or syphilitic ulceration. The existence of tuberculous disease elsewhere and, in doubtful cases, the histological examination of an excised portion of the ulcer, will clear up the diagnosis.

**Prognosis.**—The prognosis is unfavourable. It is difficult to get the ulcers to heal, and when there is advanced disease of the larynx the additional pain and interference with nutrition may hasten the fatal termination.

**Treatment.**—Rarely, when the local condition is favourable, and there is no advanced disease elsewhere, complete excision of the ulcer with suture of the wound may be possible. In other cases, after the application of eucaine, the ulcer may be scraped and treated with pure carbolic acid. Generally, palliative treatment only is feasible. To relieve pain the ulcerated surface may be painted over daily with 20-per-cent. lactic acid, followed by applications of orthoform; solutions of eucaine may also be used. Carious teeth should be extracted, and the mouth kept clean with a suitable antiseptic wash. Severe pain may call for division of the lingual nerve. The general treatment for tuberculosis should also be carried out.

PHILIP TURNER.

**TONGUE, ULCERATION OF.**—The more important forms of ulceration of the tongue are *syphilitic*, *tuberculous*, *epitheliomatous*, and *traumatic*. The first three are considered in preceding articles.

**Traumatic ulceration** is commonly caused by a carious tooth or an ill-fitting plate, and hence

## TONGUE, ULCERATION OF

is usually known as a dental ulcer. It generally occurs on the margin of the tongue opposite the tooth which has caused it. At first there is an abrasion, which, if not treated, becomes a definite painful ulcer with inflamed edges and a sloughy base. It may persist for many weeks, becoming an indolent sore with hard, firm margin. Mobility may be slightly impaired, salivation increased, and the lymphatic glands enlarged. The ulcer now resembles an epithelioma, or that form of tuberculous disease which appears as an indolent fissure at the side of the tongue. If the nature of the ulcer is doubtful, a portion should be excised for histological examination.

*Treatment* consists in the removal of the cause, the cleansing of the mouth by antiseptic washes, and some unirritating application such as glycerinum acidi borici. In recent cases this soon proves successful, but old-standing cases are intractable. In them the ulcer may be touched with chromic acid or silver nitrate, but these must be used with caution lest the irritation determine the development of epithelioma. When the nature of the ulcer is uncertain, it should be excised.

The ulcer of the *frænum* occasionally seen in children with severe whooping-cough is a variety of traumatic ulcer.

*Dyspeptic ulcers* are usually multiple. They are small, painful, and superficial, occurring on the dorsum and sides, and generally associated with similar lesions on the lips and cheeks; though they may possibly be inflamed, induration is absent. They occur in patients suffering from gastric disturbances, and treatment of the cause and the use of mouth-washes soon leads, as a rule, to their disappearance.

Ulceration occurs as a part of *mercurial stomatitis*. The ulcers are multiple and shallow, and are also seen on the gums and buccal mucous membrane. Treatment consists in the removal of the cause and the use of mouth-washes of hydrogen peroxide or potassium permanganate. Patients taking mercury should always attend carefully to the cleanliness of the mouth.

*Ulcerative stomatitis in children* may produce ulcers on the tongue as well as on the mucous membrane of the mouth. They are generally due to neglect, and clear up with antiseptic washes and general treatment.

*Herpes* of the tongue may give rise to small superficial ulcers.

PHILIP TURNER.

## TONSILLITIS, ACUTE

**TONGUE-TIE.**—In this condition the *frænum* of the tongue is so short congenitally that the tip of the organ is tied down and is not capable of protrusion. It is an exceedingly rare abnormality, although parents frequently bring their children to the doctor for this supposed defect. Often there is no abnormality at all except that the child is late in learning to speak. Should a true case of tongue-tie be met with, it is a simple matter to snip the *frænum*, thereby releasing the tongue for normal movement.

C. A. PANNETT.

**TONSILLITIS, ACUTE.**—This variety of tonsillitis occurs in four forms: (1) Simple or Catarrhal; (2) Lacunar or Follicular; (3) Parenchymatous; (4) Ulcerative.

**Etiology.**—The etiological factors are the same for the four types, and will therefore be considered together. It usually occurs between the ages of 6 and 30, and is most common in spring and autumn, and in periods of drought. One attack predisposes to another.

### Predisposing causes:

1. All conditions tending to lower the vitality of the individual, e.g. bad conditions of living, overwork, anæmia, exposure to wet and cold.
2. Septic disorders of the mouth, teeth, pharynx, and nose.
3. Frequently influenza, and less commonly scarlet fever and measles.
4. After operations on the nose or throat, when much sponging of the pharynx has had to be done.
5. When the tonsils have been incompletely removed.

### Exciting causes:

Infection with a definite organism, especially *Streptococcus pyogenes*.

**Pathology.** 1. **Simple tonsillitis.**—The mucous membrane of the tonsil becomes inflamed and the superficial vessels are dilated. The condition is usually bilateral, the mucous membrane of the nose and pharynx being frequently affected.

2. **Lacunar or follicular tonsillitis.**—This is generally bilateral, the pharyngeal mucous membrane being also involved in the inflammation. The crypts become filled with an inflammatory exudation consisting of epithelial debris, leucocytes, and micro-organisms, and present on the surface of the tonsil as yellow spots. These may coalesce, forming a false membrane.

3. **Parenchymatous tonsillitis** usually starts in one tonsil, although the other commonly

## TONSILLITIS, ACUTE

follows suit within 24-48 hours. The tonsil is much congested and inflamed; the blood-vessels become dilated, leading to an inflammatory exudate into the parenchyma and into the crypts; hence the general enlargement of the tonsil, which may project out from between the pillars.

4. **Ulcerative tonsillitis.**—This is a rarer type. There may be one or more oval shallow ulcers covered with a greyish slough. The tonsil itself is neither swollen nor greatly inflamed. Many of these cases are examples of Vincent's angina—the result of an infection with special fusiform bacilli and spirilla. There are a number of ulcers covered with dirty, whitish sloughs; these coalesce into a pseudo-membrane closely resembling that of diphtheria. (See also Vol. I., p. 148.)

**Symptoms.**—These vary to a certain extent with the type, being mildest in the simple catarrhal, and severest in the acute parenchymatous and ulcerative types. There is usually a preliminary period of malaise, with a feeling of chilliness and possibly a rigor, and there may be accompanying pains in the back, limbs, and joints. The temperature rises to 102°-104° F., the pulse becomes rapid, full, and bounding. Locally, at the outset, there is a feeling of discomfort in the throat which rapidly increases to pain, radiating up to the ear, and especially severe in swallowing. In bad cases the patient is unable to take any nourishment, the jaws become partially fixed, the head is held rigid, and the voice becomes thick and smothered.

**Diagnosis.**—In this there is little difficulty, except to distinguish the acute lacunar form, where there has been a coalescence of the exudate into one pseudo-membrane, from Vincent's angina and diphtheria. In acute lacunar tonsillitis, as against *diphtheria*, the disease is more rapid in its onset, is bilateral as a rule, is more painful, and there is a higher temperature. The urine is loaded with urates, whereas in diphtheria the urine is albuminous, and there is an absence of knee-jerks. Locally, the membrane is usually limited to the tonsillar area, whereas in diphtheria it spreads on to the pillars, soft palate, and uvula. Culture shows the presence of the Klebs-Löffler bacillus in diphtheria. In *Vincent's angina* the patient has comparatively little pain, nor is there the same general disturbance as in the other two conditions, and the disease is usually unilateral. Bacteriological examination shows the fusiform bacilli and spirilla.

**Prognosis.**—In uncomplicated cases the outlook is good, the usual period of illness being four to ten days.

**Complications.**—In bad cases the following complications may be met with:

1. *Acute otitis media* with rapid involvement of the mastoid antrum.

2. *Septicæmia*, especially infecting joints, the endocardium and pericardium.

3. *Cellulitis*, spreading into the neck and involving the submaxillary and cervical lymphatic glands.

**Treatment.**—1. **Prophylactic.**—As one attack predisposes to another, it is always advisable to establish a rigid prophylaxis. Septic conditions of mouth, teeth, and nasal cavities should be dealt with, and hypertrophied tonsils, especially if with crypts, removed.

2. **General.**—Put the patient to bed and give calomel 3-5 gr., followed in six hours by a saline. Nourishing fluids or semi-solids should be given every two hours. In regard to drugs, tinct. ferri perchlor. 2-5-min. doses in a teaspoonful of water hourly has a great repute, as also has the administration of quinine sulphate in 5-gr. doses thrice daily. If there is much pain the combination of salicylate of soda 10 gr. with phenazonum 3 gr. and caffeine cit. 2 gr. every two hours for four doses, then four-hourly, is useful.

3. **Local.**—In the very early stage a paint of menthol and guaiacol, 20 gr. of each to the ounce of sweet almond oil, is useful, as is also silver nitrate, 30 gr. to one ounce, but if after twenty-four hours the condition advances, it is better to abandon all paints. Warm applications to the neck are comforting, and the pharynx should be steamed with the following:

R̄ Menthol gr. xl.

Chlorof. ʒi.

Tr. benzoin. co. ʒi.

A teaspoonful to a pint of boiling water for inhalation.

A teaspoonful of bicarbonate of soda with two teaspoonfuls of sanitas to half a tumbler of hot water used in a spray every hour is serviceable in keeping the parts clean and gives considerable comfort to the patient. Hydrogen peroxide sprayed (warm) is also useful. When a culture from the exudate shows a preponderance of streptococci an early injection of anti-streptococcic serum is undoubtedly beneficial.

In *Vincent's angina* the part should be cleansed daily with 10-vol. hydrogen peroxide, and tincture of iodine applied.

J. GAY FRENCH.

**TONSILLITIS, CHRONIC.**—There are two types of chronic tonsillitis: (1) Chronic Hyperplastic, (2) Chronic Lacunar.

## 1. CHRONIC HYPERPLASTIC TONSILLITIS

**Etiology.**—The condition occurs chiefly in children, and has a tendency to run in families. It is almost invariably associated with the presence of adenoids. Among the exciting causes may be mentioned frequent colds and rhinitis, the exanthemata, and influenza.

**Pathology.**—There is a considerable increase in the lymphoid tissue and also in the connective-tissue stroma. Where the chief increase is in the lymphoid tissue, the tonsil becomes enlarged, soft, and friable; on the other hand, if the connective-tissue stroma is the chief object of increase, the tonsil becomes fibrous and hard. This latter type occurs in tuberculous tonsils. The writer and Dr. Ernest Shaw investigated the tonsils of 1,000 consecutive cases at the Great Northern Central Hospital, and found 3·8 per cent. of all cases to be tuberculous. The tonsils were invariably hard, small, and pale, never large and fleshy.

The enlarged tonsils may be of such a size as to meet across the middle line; or the enlargement may take place in the upper or lower parts. Various names have been applied to these various enlargements.

**Symptoms.**—As the enlarged tonsils are usually accompanied by enlarged adenoids the symptoms are those of nasal obstruction. The mouth is kept constantly open, the child is a mouth-breather, and there may be associated deafness and enlarged cervical glands. There is a special liability to colds which lead to acute inflammation of the tonsils, and enlarged tonsils predispose to attacks of scarlet fever and diphtheria.

**Diagnosis, prognosis, treatment.**—Diagnosis is usually easy, examination showing the enlarged tonsils. In adults, secondary syphilis must be differentiated. The prognosis is good with treatment which consists in removal of the tonsils. When there is a chronic enlargement of the tonsil, whether of the hyperplastic or lacunar variety, the only efficacious treatment is total removal. This is done in one of two ways—(1) dissection enucleation, (2) enucleation by guillotine. The latter method is the one in general use and answers admirably; the former should be reserved for cases of deeply embedded tonsils, and when there has been a previous operation which has left the bases of the tonsils behind. The operations

are considered in more detail under **ADENOIDS AND TONSILS, OPERATION FOR REMOVAL OF.**

The chief danger in removal is hæmorrhage, which is much more prone to occur in an adult than in a child. The best methods of preventing and dealing with it are:

(1) *Proper preparation of the patient.*—Adults should be given calcium lactate in 10-gr. doses thrice daily for a week previous to operation.

(2) *Never operate on an inflamed tonsil.*—This should be locally treated first. A paint of resorcin 40 gr. to an ounce of glycerin has been recommended for local application for some days before removal.

(3) *Do not use too sharp a guillotine.*—A blunt instrument is much safer.

If hæmorrhage occurs—

(1) Apply direct pressure over the bleeding area with a gauze swab soaked in adrenalin 1 in 1,000.

(2) Give morphine  $\frac{1}{2}$  gr., with  $\frac{1}{10}$  gr. ergotin, hypodermically to an adult.

(3) If the hæmorrhage still continues, put a packing into the cavity between the pillars and stitch these over it. The packing must be removed in 24–48 hours.

(4) A hypodermic injection of horse- or ox-serum is useful.

## 2. CHRONIC LACUNAR TONSILLITIS

A chronic inflammation of the tonsil accompanied by accumulation of caseous plugs in the lacunæ.

**Etiology.**—The condition usually follows a series of attacks of the acute variety, and is also seen in connexion with peritonsillitis and in septic conditions of the mouth and nasopharyngeal regions.

**Pathology.**—The chief changes occur in the crypts of the tonsils, which in themselves may not be enlarged. These crypts are filled with a secretion which on inspection presents whitish-yellow points. The secretion consists of desquamated epithelium, cholesterol, leucocytes, and micro-organisms. There is, as a rule, little surrounding inflammation.

**Diagnosis.**—The only condition to be differentiated is *keratosis*. In this the secretion points are not confined to the tonsils, and are firmer, whiter, and more difficult to remove.

**Symptoms.**—The patient complains of a foul odour and taste, with a sensation of roughness and irritation of the tonsil, and occasional pain, especially on swallowing.

**Treatment.**—The best treatment is total removal of the tonsil. Should this be refused,

## TORTICOLLIS AND WRY-NECK

the tonsil may be cocaineized by spraying with 10-per-cent. solution of the drug. Then clear out each crypt and inject into it a 10-per-cent. solution of silver nitrate, taking care not to allow any of this solution to run down the throat.

J. GAY FRENCH.

**TOOTHACHE** (*see* DENTAL PAIN).

### **TORTICOLLIS AND WRY-NECK.** —

There are several widely differing pathological processes which, in their incidence on the muscles of the neck, produce clinical conditions not seldom classed together unthinkingly as spasmodic torticollis; but successful treatment depends on the clear recognition of the different underlying morbid agents producing the conditions. Some cases of torticollis are undoubtedly benefited by operation, in others operation fails simply because the physician in charge of the case has failed to recognize the pathogenic basis of the affection.

The following **classification**, adapted from Cruchet, will be found of practical value:—

(1) *Neuralgic torticollis*.—Corresponding to the involuntary facial movements of trigeminal neuralgia—so-called *tic douloureux*—are involuntary neck movements in occipital neuralgia—*neuralgic torticollis*. With the onset of pain the neck movements appear; they are at a maximum in paroxysms of pain, and they disappear with the temporary subsidence of the neuralgia. The torticollis movements are either tonic or clonic; they are certainly often slow, and inclined to be mainly tonic.

(2) *"Professional" torticollis*.—This form of torticollis—an occupation neurosis has been rather lost sight of, but it deserves mention; it exactly resembles writer's cramp, and is in some instances actually associated with that condition. In a typical case the involuntary movements occur solely at the moment of execution of a given functional or occupational act. It is said to be noticed mainly in cobblers, tailors, and paviours, but it has been seen in a number of other employments. Both the neuralgic and the occupational variety may persist as a spasmodic torticollis though the original cause be removed.

(3) *Paralytic torticollis*.—Just as facial spasm may arise secondarily to a peripheral facial palsy, so torticollis, a spasm of the neck muscles, may be secondary to a paralysis of those muscles. As a rule, following on a chill or exposure, the patient complains of a "stiff neck," and a little later notes that his head

is tilted to one side. This attitude-torticollis becomes more pronounced when he is on his feet or walking, as the neck muscles then have to support the head and any asymmetry is noticeable. Later still, spasmodic torticollis may develop on this prepared site.

(4) *True spasmodic torticollis*.—This variety is divided into idiopathic and symptomatic sub-varieties. The former is comparable to facial hemispasm, and its pathogeny is similarly obscure. The lesion is possibly in the peripheral nerves, and its nature neuritic. This variety is familiar. The contractions are tonic, clonic, or tonico-clonic; they vary in intensity and duration and in the degree of accompanying pain. Associated movements are common. The influence of the will on this condition is, as a rule, minimal, or nil. In the symptomatic sub-variety the torticollis occurs in the course of, or as a sequel to, influenza, rheumatism, typhoid, diphtheria, etc. It may be an epileptic equivalent.

(5) *Rhythmic torticollis* is that form which occurs in the course of spasms nutans and analogous conditions, and is therefore a symptomatic variety. Under this heading are included rhythmical movements in idiots and epileptics, the rhythmical to-and-fro tremors of the head in hysteria, etc.

(6) *Tics of the head and neck, and so-called mental torticollis*. Many cases of torticollis are obviously of the nature of tic (*see* TICS AND HABIT-SPASMS).

The **treatment** of *neuralgic torticollis* should be directed primarily to the neuralgic side. Hence recourse should be had to the remedies of proved value in neuralgia. Just as *tic douloureux* disappears when the concomitant facial neuralgia is relieved, so with the torticollis movements of occipital neuralgia. Any of the great collection of serviceable anodynes may be utilized. Local applications of sedative galvanism, sedative massage, liniments, lotions, counter-irritants, may be of value. Alcoholic injections at painful points over the occipital nerves may have to be employed.

As for *occupational torticollis*, the patient must give up the work which necessitates the movements concerned, as a preliminary to further treatment. This is essentially the same as in occupation neurosis (*see* NEUROSES, OCCUPATION).

In *paralytic torticollis*, sedative galvanism may be found advantageous.

For true *spasmodic torticollis*, massage to the weaker neck muscles, galvanism, and re-



## TRANSFUSION

sistive exercises to strengthen them may be recommended. Sedatives are sometimes of value, but oftener they completely fail. Rest in bed, with the head immobilized between sandbags, or with the chin and head weighted by an extension apparatus, may be tried. Sometimes suspension is distinctly useful. Alcoholic injection of the motor nerves concerned might conceivably help, but I am not aware whether this suggestion has ever been acted on. In the last resort a complete Keene-Stirling operation—section of the sterno-mastoid nerve supply on one side and of the posterior primary divisions of the upper three or four cervical nerves on the other—may be necessary. In some cases the result is undoubtedly good.

The treatment of *symptomatic spasmodic torticollis* and of *rhythmical torticollis* is that of the underlying condition.

For treatment of *torticollis-tic*, see TICS AND HABIT-SPASMS. S. A. KINNIER WILSON.

**TOXINS** (see PATHOLOGY, CHEMICAL, MODERN DEVELOPMENTS OF).

**TRACHOMA** (see CONJUNCTIVITIS).

**TRANSFUSION.**—Transfusion of blood has received a good deal of attention during the last decade and was extensively used with satisfactory results during the War. It has most frequently been employed for anæmia resulting from severe hæmorrhage, but it is also sometimes beneficial in other varieties of anæmia, especially in pernicious anæmia. Other indications are shock, spontaneous hæmorrhage (e.g. the hæmorrhagic disease of the new-born, purpura), toxæmias, especially those of pregnancy, and acute infections.

The effect of transfusion is two-fold—replacement of blood and stimulation of bone-marrow. The first of these is clearly of greater consequence when transfusion is employed for loss of blood from hæmorrhage; and provision of missing blood elements may play a part, too, when certain spontaneous hæmorrhages are being combated. But the red cells transfused remain at most for no more than ten days, so that in those anæmias where blood is being rapidly hæmolyzed or inadequately produced, no lasting good results can be expected unless reaction of the bone-marrow follows. In toxæmias and infections the rationale of transfusion may be the injection of a healthy or immune serum, and may thus be of the nature of passive immunization. In shock the object

aimed at is the increase in the amount of the circulating blood.

**Selection of the donor.**—Whatever the method employed, the donor must be selected carefully. He must be healthy, his blood should be examined for blood parasites, notably malaria, and if time permits, a negative Wassermann reaction should be obtained. The serum of the donor should not agglutinate the corpuscles of the patient, nor should the serum of the patient agglutinate the corpuscles of the donor, and only if the two bloods are found to be compatible should transfusion be proceeded with. (For the grouping of bloods in this regard, and tests to be employed, see SEROLOGICAL DIAGNOSIS, p. 184.) In cases of emergency, when the testing is impracticable, a small preliminary transfusion should be done half an hour before, so that any gross incompatibility may be recognized.

**Methods.**—The methods which have been employed are thus classified by Stansfeld:—

1. Direct: By anastomosis of vessels, with or without a cannula.
2. Indirect:
  - i. By the use of a syringe.
    - (a) Syringe transferred from needle in vein of donor to needle in vein of recipient.
    - (b) Syringe with two-way tap leading to veins of donor and recipient.
  - ii. By the use of a receiver.
    - (a) With paraffin lining.
    - (b) With the addition of an anti-coagulant.

1. The **direct method** has the advantage that the danger of clotting is slight and the blood is not exposed to the air, but it requires considerable surgical skill and the amount transfused cannot be determined. Moreover, it is not practicable if several transfusions are required. It is becoming largely superseded by indirect transfusion.

2. **Indirect method.**—i. The needles in the syringe method may be replaced by cannulæ. In the better of the two methods a needle or cannula is inserted into the median cephalic or median basilic vein of the donor and another into the corresponding vein of the recipient, and blood is transferred from one to the other by record syringes of 20 c.c. capacity worked in series. The syringes and cannulæ or needles are previously sterilized and lubricated by drawing liquid paraffin through them, and between successive injections a small amount of normal saline is forced through the needles or

## TRANSFUSION

cannulæ to wash them free of blood. This method is a good one, but requires two operators, assistants, and a large number of expensive syringes.

ii. (a) The paraffined-glass container which has been most used is the Kimpton tube; it requires elaborate precautions, coagulation is apt to occur, and there appear to be no advantages over the method about to be described.

(b) The anticoagulant commonly employed is citrate of soda; its introduction has rendered blood transfusion a comparatively simple procedure. Into a sterilized glass receptacle graduated to 500 c.c. are introduced about 60 c.c. of sterile 2-per-cent. sodium citrate solution in normal saline. After the donor's arm has been constricted a hollow needle is inserted into an elbow vein, and the blood is allowed to flow into the citrate solution, which is stirred with a glass rod to ensure thorough mixing, until the 500 c.c. mark is reached. The citrated blood is then strained through sterile gauze to remove the froth and introduced slowly into the recipient's vein by an ordinary saline infusion apparatus. The container may be kept warm by standing it in warm water. If smaller amounts of blood are to be transfused the proportion is easily adjusted; thus if 250 c.c. is the amount decided upon, only 30 c.c. of the citrate solution is used, and blood is run into the container up to the 250 c.c. mark. If more than 500 c.c. is to be used a second receiver should be employed, the first standing in warm water until required.

**Dosage.**—The optimum dosage has still to be determined, but where stimulation of bone-marrow is the end in view, several small transfusions of say 250 c.c. are probably to be preferred to one larger one. In hæmorrhage larger amounts comparable to the amount lost are indicated. In cases of spontaneous hæmorrhage such small quantities as 20 c.c. have proved beneficial.

Most of the ill-effects recorded are ascribable to isohæmolysis, but, even when the donor has been scrupulously attended to, febrile reactions occur in about 25 per cent. of transfusions and rigors in about 10 per cent. Giddiness, buzzing in the head, cramps, vomiting, faintness, and pain in the back sometimes follow.

FREDERICK LANGMEAD.

**TRANSILLUMINATION** (see SINUSES, ACCESSORY AIR, DISEASES OF, p. 200).

**TRAUMATIC HYSTERIA** (see NEUROSES, TRAUMATIC).

## TRENCH FEVER

**TRAUMATIC INSANITY** (see CONFUSIONAL INSANITY).

**TRAUMATIC NEUROSES** (see NEUROSES, TRAUMATIC).

**TREMOR** (see ATHETOSIS AND TREMOR).

**TRENCH FEVER.**—A specific infectious fever characterized by pains, a peculiar type of pyrexia, frequent splenic enlargement, rash, prolonged course and slow convalescence. The disease was first recognized among troops in the Great War.

The first cases in the British Army were described among front line troops in France in 1915. Afterwards the disease became very prevalent throughout the Army areas in France and Belgium, and made its appearance to a far smaller extent among the troops of the Eastern Expeditionary Forces. Cases have also occurred among the civilian population.

**Etiology.**—Early experiments demonstrated that the blood of a patient suffering from the disease in an acute stage is infective. Dried urinary deposits have been proved to convey infection, and the sputum has been shown to be infective also. Two types of organisms have been described, but confirmatory evidence is lacking. The organisms described are: (a) *Spirochetes*. These have been demonstrated by Coles in blood-films from experimental cases on the second day of the illness. The presence of spirochætes in carefully collected urines, especially about the fifteenth day of the disease, has also been recorded. (b) A filter-passing *anaerobic organism* has been isolated from the blood, inoculation experiments with cultures of which have reproduced the disease in the human subject.

The chief vehicle of infection has been shown conclusively to be the body-louse. The virus is present in the alimentary tract of the infected louse, and can readily be conveyed by rubbing the crushed bodies of such lice, or their excreta, into the scarified skin. The bite of the infected louse is non-infective. The infection may also be conveyed by *carriers*, for the blood of individuals during the chronic stage of the disease has been shown to be infective many weeks after the onset of the original illness.

**Symptomatology.**—The *onset* is sudden, with headache, general malaise, prostration and sometimes fainting. Initial rigor is uncommon, and catarrhal symptoms are absent. Vomiting and diarrhœa may usher in the disease and persist for two or three days, and if there is

## TRENCH FEVER

great abdominal pain and tenderness, appendicitis may be simulated. Cerebro-spinal symptoms occur in a small proportion of cases, and the classical picture of meningitis may be accurately copied. Lumbar puncture reveals slight rise in the pressure of the cerebro-spinal fluid, but no other abnormality.

**Fever.**—Two main types can clearly be recognized—the long and the short, but intermediate forms are common. Inoculation experiments have shown that the types do not “breed true,” but that the blood of a patient suffering from the long type may produce the short type of the disease in another individual, and vice versa.

The *long* type presents the more striking picture; the temperature rises rapidly to 103° or 104° F., remains about this level for twelve or sometimes twenty-four hours, and then falls rapidly; by the morning of the third day it reaches normal. Thereafter the chart is made up of a succession of peaks, whose bases lie below the “normal” line, and extend over a period of three days each, and whose summits occur at five-day intervals. Variation from this may occur; the intervals may be longer or shorter. In the later stages such variation is the rule, but in the classical case a series of four or five peaks appears at regular five-day intervals between each peak with a tendency to decrease in height throughout the series. These are followed by an irregular pyrexia which is described under Chronic Trench Fever (p. 357). The *short* type presents a picture composed of a rapid rise, a course sustained with little variation for three to five days, a rapid fall, and absence of regular recurrence. The febrile stage may be much longer, even ten or twelve days. When it is shorter other conditions, notably infections from the alimentary tract, may be incorrectly classed in this group.

**Pain.**—The early headache, backache and limb pains of the onset are of short duration. They are followed later by the pains which are characteristic of the disease, and commence at the time of the second or third rise of temperature in the long type—after defervescence in the short. The pains are situated where large bones are subcutaneous, e.g. shin, knees, ankles, elbows, ulna, or scapular spine; they occur also in the large muscles of the thigh and loin. The hands and feet are not affected, and the flexor surfaces of the limbs, with the exception of the popliteal space, usually escape. The pains are intermittent, of gradual onset and slow relief, invariably worse at night, and by

day may be absent. They recur in bouts lasting two or three nights, with intervals of a few days. They are described as “burning,” “tearing,” or “throbbing,” and may be associated with such great superficial tenderness that the pressure of light bedclothes on the shins may be unbearable.

The pain commences usually about 6 p.m., gets steadily worse till midnight, then slowly diminishes, and the patient is able to sleep. Next morning the pain has completely ceased, to recommence in the early evening.

**Alimentary system.**—The tongue presents no constant feature, but a peculiar yellow glaze is seen in some cases during the febrile stage and at the time of each relapse. Vomiting and diarrhoea may occur at the onset of the disease. Discomfort or even pain may be felt in the hypochondriac regions, but no enlargement of the liver can be detected.

**Circulatory system.**—One of the outstanding features of the disease is the slowness of the pulse in the acute stages. During the first few days the pulse-rate is increased in proportion to the rise of temperature. The slowing appears usually at the time of the second relapse. It is not unusual to find a pulse-rate of 90, with a temperature of 104° F. Often, especially in the apyrexial periods, the pulse-rate falls to 60, sometimes even lower. In the later stages this slowing of the pulse-rate is replaced by an irritable quickening which may persist for several months.

Slight changes are found in the blood. In the early stages there is a slight increase in large mononuclear cells and eosinophils. In the later stages slight secondary anaemia is common. Myocardial derangement is frequent, but pericarditis and endocarditis do not occur. Complaints of chest-pain are rare during the acute stage of the disease.

**The spleen.**—Some enlargement is the rule. It occurred in 85 per cent. of a series of experimental cases, but ordinary clinical records do not yield such a high proportion. The enlargement is not constant in any given case; it may vary from day to day, and there is a tendency for the splenic volume to wax and wane with the rise and fall of the temperature curve. In many cases the enlargement can only be detected by a series of careful examinations throughout the disease. When palpable the organ is firm and not tender. The enlargement may be recognized as early as the fifth day, and as late as the seventh week.

## TRENCH FEVER

**Respiratory system.**—Trench fever does not affect the respiratory system. Should antecedent or coincident bronchitis be present, infection may be conveyed by means of the sputum.

**Renal system.**—The coincidence of the pyrexia, shin-pains, and slow pulse of trench fever, with the oedema, breathlessness, severe albuminuria or hæmaturia of acute nephritis is occasionally seen. The majority of trench-fever patients present no renal symptoms, but a considerable degree of albuminuria is not uncommon during the febrile stage. The infectivity of the urine and the presence of spirochætes have been alluded to. It is probable that the infective agent may damage the kidney during its excretion. When renal and trench-fever symptoms occur together the renal are usually the more striking, and in many cases the trench fever is a relapse or a recurrence after a previous and often a mild attack.

**The skin.**—Sweating is usual, often profuse. Rashes are common, and have been observed much more frequently in experimental cases than in others; one reason for this is the difficulty in recognizing slight rashes as a new phenomenon unless the skin has been known by careful preliminary observation to be healthy before the onset of the disease. Erythematous, macular, and papular forms have been described. The papular form is the most common; it occurs as small pink papules, not sharply defined from the surrounding skin, situated chiefly on the trunk and most often about the lower thorax and upper abdomen. The spots never occur on the face, even when the rash is profuse elsewhere. They come out early in the disease, sometimes as early as the second day, each spot lasting for twenty-four to forty-eight hours; they appear in successive crops, and may be found at any time during the first four weeks. Urticaria is seen occasionally. Herpes is very rare.

**Nervous system.**—Symptoms referable to the nervous system may occur at any stage of the disease. The meningeal type of onset has already been referred to. During the height of the disease and its convalescence, less definite symptoms are common. Headache, usually frontal, but sometimes occipital, is a common complaint, and its onset frequently heralds a recurrence of pyrexia. Sleeplessness is a serious and exhausting symptom. While the disease is at its height, this is due to pain and is relieved spontaneously when the pain passes off,

but during convalescence it is occasionally troublesome in spite of the absence of pain.

Numbness and tingling of one or more limbs is often complained of. Hyperalgesia in the later stages is common, especially in the areas of the 8th cervical, 1st dorsal, 7th and 8th dorsal, and all the lumbar segments; it may persist for many weeks (Carmalt-Jones). Analgesia and anæsthesia in irregular areas about the legs and shoulders is very common in late stages. When cases are seen first at this stage the symptom may be of diagnostic importance.

**Chronic trench fever.**—In a large proportion of cases recovery from an initial attack of trench fever is incomplete. The disease passes into a chronic form which may persist for many months. This tendency appears to be as great in cases which were originally mild as in those which were severe. Its recognition is important, for it affords a clue to the diagnosis of many otherwise obscure disabilities, and in army life has been responsible for much sickness and invalidism. The manifestations of chronic trench fever fall into three main groups: cachectic, cardiac, myalgic. In the *cachectic* form the patient is anæmic, languid, sleeps badly, easily becomes breathless, has "rheumatic" pains, and may show areas of hyperalgesia or analgesia. The *cardiac* form presents the familiar signs and symptoms of an irritable and tired heart—præcordial or subscapular pain, tenderness of skin or muscle in corresponding areas, breathlessness, and instability of pulse-rate. The *myalgic* form combines cachectic symptoms with severe recurrent pains in the limbs, brought on by exercise or exposure. In the recognition of chronic trench fever the following points are important:

1. A history of a previous acute attack, sometimes mild and neglected.

2. Temperature course. Accurate, regular and frequent records will reveal a "swinging" but subnormal temperature. At times the apex of the swing may rise for a few hours above the normal line, but usually remains subnormal, and unless accurately recorded, its characteristic appearance of a "pus temperature below the line" is missed.

3. The character of the pain, its transient nature and nocturnal regularity.

4. Paræsthesia and analgesia are very common many weeks after the original attack.

5. Moderate enlargement of the spleen, which can often be detected by regular or repeated examinations, is very important. It

## TRENCH FOOT

may still occur several weeks after the original attack has apparently passed off.

**Diagnosis.**—The typical attack of trench fever is so distinctive as to be self-evident. In the less typical forms the disease may in some particulars resemble malaria, relapsing fever, undulant (Malta) fever, or paratyphoid. The three former can be recognized by blood-examination; the distinction between trench fever and paratyphoid is sometimes very difficult. The important points are indicated in the following table:—

	TRENCH FEVER	PARATYPHOID
<i>Onset</i>	Sudden	Usually gradual.
<i>Rash</i>	Irregular, early or late	Appears on the seventh day
<i>Spleen</i>	Slight periodic enlargement	Greater single enlargement
<i>Relapse</i>	Sudden, repeated	Gradual, single or absent
<i>Tongue</i>	Not characteristic	Usually typical
<i>Widal</i>	Negative	Positive after seventh day
<i>Blood-culture</i>	Sterile	Specific organism during first week.

**Treatment.**—No specific treatment is yet available. In the acute stages organic compounds of arsenic, colloidal metals, pharmacopœial and proprietary drugs have all been tried: the reports on their results are conflicting and disappointing. Symptomatic treatment has to be substituted. In the acute stage relief is afforded by phenacetin (5 gr.) combined with caffeine citrate (5 gr.); pulv. ipecac. co. (15 gr.); tr. opii (15 min.) at 6 P.M., or omnopon ( $\frac{1}{2}$  c.c.) at 8 P.M. Sodium salicylate has no value; aspirin has some effect. In the later stages the pains are often relieved by a mixture containing vinum colchici (10 min.) and pot. iod. (2 gr.), given three times a day for one or two weeks. Local applications, as hot or cold compresses, lot. plumbi evaporans, lin. A.B.C., or ung. menthol, sometimes give relief.

Cardiac disabilities respond to graduated exercises, but till the temperature record becomes steady the cause of the embarrassment must be considered to be still at work, and exercises are not indicated. Digitalis is unnecessary.

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**TRENCH FOOT.**—A circulatory embarrassment of the lower extremities, which may proceed to stasis and gangrene of the feet.

**Etiology.**—This condition was very common in the early stages of the late War,

and though it was not unknown in previous wars—for example, the Crimean campaign—it was not so frequently seen. Three factors are prominent in determining the onset—cold, damp, and pressure. Cold alone does not seem to be a sufficient cause. With extremely low temperatures frostbite occurs, but men who suffer from trench feet have usually not been exposed to a temperature so low as the freezing-point. Wet, muddy trenches, not frozen ground, conduce to this disorder. Men who move about freely are much less likely to suffer than the immobilized soldier on sentry duty. Constriction of the legs plays its part, and it is worthy of note that ordinary puttees contract to a considerable extent when they become wet, and so have a strangulating effect upon the circulation.

Trench foot is not confined to war; it is seen now and then in civil practice, and not only in vagrants who have lain out all night.

**Pathology.**—The pathology is essentially that of frostbite, and the process has been studied experimentally in animals. The arteriole and capillary walls bear the brunt of the damage at first; they become more permeable, and a fibrinous exudation is seen lining their walls and external to them. Necrosis of the tissue-cells results.

**Symptoms.**—The onset is gradual and the effect of the causal factors seems to some extent to be cumulative. Thus, during one stay in the trenches a man may suffer from some numbness and pallidity of his toes, from which he recovers completely when his unit is withdrawn from the line; yet so soon as he returns to the trenches the condition reasserts itself, and now his feet appear to be more vulnerable to the baneful influences. After the numbness and pallor have lasted for a short time in one or more toes, swelling comes on. Then the affected parts assume a red, blotchy appearance. The circulation is obviously sluggish. As in other varieties of gangrene, the colour varies from purple, green, blue, to black. Long before this stage, however, the patient complains of severe pain in his toes, so that very often sleep is much interfered with.

**Course.**—Recovery may take place when the pathological process has not proceeded to the actual death of the tissues. The redness gradually goes, the swelling slowly subsides, and the foot returns to a normal state, though pain may be felt long after apparent objective recovery. Gangrene, when it occurs, may be of any degree. In the superficial forms

## TRENCH FOOT

blisters appear, and, breaking, leave ulcers which do not reach through the deep fascia. These sluggish ulcers, under appropriate treatment, will heal. In the more severe cases whole toes or even the anterior part of the foot (rarely more) die. The gangrene is usually dry, but may be of the moist variety. Two very serious complications which may arise must always be borne in mind—tetanus and gas gangrene.

**Prevention.**—Care of the feet is most important. They should not be allowed to become wet. The boots should be waterproof and of ample size, so that two pairs of socks may be worn without subjecting the feet to dangerous pressure. By some it is recommended that between the two pairs of socks a third thin pair, made of oil silk, should be worn. Ordinary cleanliness of the feet is important, and rubbing of the feet daily, when there is the opportunity, is a good prophylactic routine to insist upon. Nothing should be allowed to constrict the leg, and puttees might well be replaced by a simple legging which would allow more play to the muscles. In the trenches movement may avert the onset. It is the man who stands for prolonged periods in the mud who becomes affected.

**Treatment.**—When the disease has made its appearance, great care must be taken not to increase the trouble by ill-advised treatment. Warmth must be applied to the parts very gradually. Indeed, at first an evaporating lotion is useful. The patient should be warmly wrapped up. Gradually tepid water may be used for bathing, and then warm water. This stage having been reached, daily massage with warm oil is indicated, or the paraffin treatment may be adopted. Every day for half an hour the feet are kept in a molten paraffin bath at 60° C. The melting-point of the paraffin should be 45° C. Afterwards the feet are wrapped up in a layer of wool.

If ulcers are present, every antiseptic precaution should be taken. The whole foot should be scrupulously cleansed and enfolded in sterile gauze. A bath of 1-per-cent. picric acid is well spoken of by some surgeons. Ulcers and granulating surfaces should be dealt with as described in WOUNDS, TREATMENT OF, and ULCERATION. The paraffin treatment, as described in BURNS AND SCALDS, is also an effective method of treatment.

When gangrene occurs the ordinary rules for the treatment of this condition hold good. Frequently only single toes need be amputated.

## TRICHINELLIASIS

When the front part of the foot is dead the best amputation is that of Syme. Only when the gangrene is moist are higher amputations indicated.

Every man with trench foot should receive prophylactic injections of antitetanic serum.

C. A. PANNETT.

**TRENCH NEPHRITIS** (*see* NEPHRITIS).

**TRICHIASIS** (*see* EYELIDS, AFFECTIONS OF).

**TRICHINELLIASIS.**—The disease caused by infection with the parasitic worm, *Trichinella* (*Trichina*) *spiralis* (Owen).

**Life-history of the parasite.**—The parasite passes all three stages of its life-history, as adult, embryo, and encysted larva, in the human body. The worms are ingested in the larval condition; their cyst-walls are dissolved by the gastric juice, and the liberated larvæ pass into the intestine. Here rapid growth takes place, and sexual maturity is attained in about two days. Both the sexes are long and cylindrical, the male being 1.5 mm., the female 1.6 to 1.8 mm. in length. The anterior end in both sexes is pointed, and provided with a small punctiform mouth; the posterior end in the male terminates bluntly and is provided with two clasping organs, the penis being formed by eversion of the cloaca; in the female the posterior end is rounded. (Fig. 96.)

At this stage copulation takes place and the male dies, but the female rapidly increases in size, reaching a length of 3 to 4 mm. Growth is due chiefly to development of the ova, which soon become embryos and fill the oviducts

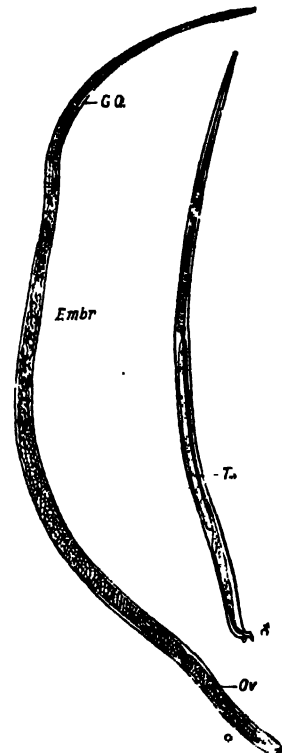


Fig. 96.  
*Trichinella spiralis*.  $\times 45$ .  
(After Brumpt.)  
G.O., Genital opening; Embr., embryo; Ov, ovary; T., testes.

## TRICHINELLIASIS

in four or five days after fertilization. The young worms escape by the genital opening, which is situated on the ventral aspect of the worm about one-fifth of the distance from the head to the tail. Embryos to the number of about 1,500 escape in gradually diminishing quantity for six or seven weeks, and then the adult female dies. The embryos have a thick, rounded anterior extremity and taper to a fine point at the posterior end. They are 90 to 100  $\mu$  in length by 0.6  $\mu$  in width.

The minute worms at once make their way to the muscles of their host. Reaching the lymphatic circulation, they proceed to all parts of the body, many passing into the pleural, peritoneal, and pericardial cavities in search of striated muscle. They may wander for ten days before settling down, and during their journey they grow and develop to some extent.

Once in a muscle, they push their way into the interior of a muscle-fibre, and then begins the stage of encystment. The cyst is formed in part of chitin secreted by the parasite, and is in part due to the inflammatory reaction of the tissue of the host by which new connective tissue is formed. By continuous rotatory movements of the coiled larva the cyst is moulded to its final shape, which is like that of a lemon, oval with pointed extremities. The encysted larva, about 1 mm. in length and 0.04 mm. in width, lies coiled spirally in the albuminous fluid contents of the cyst. The cyst itself is about 0.4 mm. long by 0.25 mm. broad. Occasionally two or even more larvae are found in a single larger cyst. Complete encystment takes place about 18 days after infection, and at this stage, if the cyst reach the stomach of another animal, the worm can develop to sexual maturity in its new host. Unless this takes place the larva becomes quite quiet and can live without further change for a very long period, even twenty or thirty years, at the end of which time it is still capable of development. As time passes the cyst gradually becomes thicker, and calcification may take place.

The proper host of the trichinella is undoubtedly the rat, and in Germany and the United States of America 8 to 10 per cent. of these animals harbour the parasite. The trichinella can develop in many other mammals besides rats, the most important from the human standpoint being the pig. Pigs become infected by eating pork and swine offal containing cysts, and also by eating dead rats.

Rats, besides destroying and eating one another, also eat pieces of pork and offal given to the pigs. The highest incidence of trichinelliasis thus occurs where this method of feeding pigs is prevalent.

**Etiology.**—Infection in man is caused by eating some article of food made from portions of a pig containing the encysted larva, a condition in which the parasite is most resistant. Decomposition of the meat, drying (smoking) and pickling, high temperature and low temperature often fail to kill it. The larvae can resist a temperature of  $-20^{\circ}\text{C.}$  or  $+70^{\circ}\text{C.}$  The latter temperature is not reached in the centre of a ham after six hours' boiling, so that though thorough cooking will destroy many parasites, it cannot be relied upon entirely to prevent infection.

Another point of importance is that some parts of the pig are especially likely to contain cysts. These are the diaphragm, muscles of the neck and eye, the intercostals, and, of the large muscles, the parts nearest the tendons. Cysts also occur in the connective tissues and in the wall of the intestine. Such parts are all largely used in some countries to make up into sausages, whereas in others they are discarded. The habit of eating pickled or smoked pork with little or no cooking is also a very important factor in human infection. In man the disease is most often met with in Germany, North America, Holland, Denmark, and Scandinavia. It is rare in Great Britain and France.

**Symptoms.**—The symptoms of trichinelliasis may roughly be divided into three stages, corresponding to the stages in the life-history of the parasite: (1) The initial or gastro-intestinal stage. (2) The stage of general infection, in which myositis is a prominent feature. (3) The stage of regression (encystment of embryos).

Differences in the symptoms depend very greatly on the severity of the infection. Slight cases occur with symptoms so vague as to be unrecognizable except in connexion with a definite outbreak of the disease. In the absence of this aid to diagnosis they are mistaken for dyspepsia, muscular rheumatism or even true rheumatism, the latter mistake arising because the trichinellæ are prone to attack the tendinous ends of muscles.

**First stage.**—In the more severe cases the symptoms are pronounced, and in some respects very characteristic. If many cysts have been swallowed, symptoms begin in twelve to twenty-four hours. There are vomiting, diarrhoea,

## TRICHINELLIASIS

paroxysms of severe abdominal pain, cramp in the limbs, and collapse with cold extremities. The tongue becomes furred and the breath offensive. The onset may be so acute as to cause a suspicion of cholera, and death occasionally occurs quite rapidly from collapse. In other cases symptoms are much less severe, and there are merely a furred tongue, loss of appetite, nausea, and diarrhoea or constipation. The intestinal symptoms continue alone for the first nine or ten days, and after that are accompanied by those of the second stage, but they continue for several weeks, the diarrhoea finally giving place to an obstinate constipation. Two features of some diagnostic value occur, sometimes before the second stage is established. The first is a transient œdema of the eyelids, with or without chemosis, which begins on the sixth to the eighth day, and lasts from three to five days, and the second is pain in the muscles, which precedes the invasion by the embryos.

**Second stage.**—About the fifth day, pains due to actual muscular infection begin, and steadily become worse. With the myositis there is profuse sweating, often accompanied by a miliary eruption and followed later by the formation of pustules. The urine becomes diminished. Insomnia, delirium, and mental hebetude are prominent, so that the case may resemble typhoid. Fever, which begins in the first stage, often continues for three, five, or even six weeks. It is remittent or intermittent, with maxima of 104° to 105° F.

About the fourth or fifth week a second œdema appears and affects 90 per cent. of the cases of moderate or considerable severity. It appears in the face, neck, limbs, and trunk, and may be extreme in degree. Polyuria may be present, but in no case is there albuminuria.

The muscles themselves become swollen, hard, and tender. Movement may become so agonizing that the patient lies motionless, curled up with all the limbs partially flexed. The neck is stiff and, of the limb muscles, the biceps and gastrocnemius are especially affected. Mastication, speech, and eye movements are very painful, and the muscles may be so inflamed as to cause aphonia and fixation of the eyeballs. The diaphragm and intercostals are generally the seat of severe myositis, and this is a source of danger to life. Respiration becomes difficult, dyspnoea with attacks like asthma supervening. Bronchitis and hypostatic congestion of the lungs are common.

Death may take place from asphyxia due to myositis or from the pulmonary complications.

**Third stage.**—The third stage is one of gradual subsidence of all the symptoms, due to death of the adult trichinella and encystment of the embryos. Wasting and a cachectic condition are very noticeable at this period. At the end of six or seven weeks, convalescence is usually established. In very favourable cases it may begin at the end of the third week, or may, on the contrary, in bad ones be protracted to the eighth or twelfth. Sometimes for years after an attack there may be muscular pains.

**Blood changes.**—The red corpuscles are unaffected, but there is a leucocytosis often proportionate to the severity of the illness. In the very mild cases no increase is observed, but in average cases the leucocytes number about 15,000 per c.mm., and often more in the worst cases. With this increase in leucocytes there is an increase in the percentage of eosinophils. The eosinophilia varies from 10 to 80 per cent., averaging about 30 per cent., and its intensity is a rough index of the degree of infection. These blood changes begin soon after the entrance of the parasites into the body, and continue until all the embryos are encapsulated. Positive results can thus be obtained from the blood picture both early and late in the illness.

**Diagnosis.**—As soon as the presence of an epidemic is established, cases of the disease should be recognized with comparative ease; but isolated cases are by no means easy to recognize early, even when the disease is prevalent, and extremely difficult when it is rare.

Those with a very acute onset may resemble *cholera*, for though *cholera* is a tropical disease and trichinelliasis a disease of the temperate zone, their ranges overlap.

Later on trichinelliasis is very like *typhoid fever*, but there is an absence of the headache so characteristic of the onset of the latter disease. The stiff-neck, gastro-intestinal and mental symptoms may simulate it closely. Rose spots and enlargement of the spleen are absent, however, and the Widal reaction is negative. The muscular pains are too severe for typhoid fever, and the early facial œdema is a valuable distinguishing feature. The blood pictures are quite different, even at an early stage, leucopenia occurring in typhoid, and leucocytosis with eosinophilia in trichinelliasis.

From *acute rheumatism* the disease should be rapidly separated by the fact that the swelling



## TRICHINELLIASIS

and tenderness are not localized to the articular and periarticular tissues.

From *beriberi* it may be distinguished by the retention of the knee-jerks, and absence of anæsthesia and of cardiac dilatation with murmurs. Both diseases may occur in groups of cases, and in both there is extensive œdema with muscular pains, tenderness, and dyspnoea.

From *acute nephritis* the absence of blood, albumin, and casts in the urine is a sufficient differentiation.

In doubtful cases, worms may be found in the stools, and a small piece of the biceps near the lower end may be examined, after removal with a harpoon or excision with a scalpel, for encysted embryos. Pieces of the food suspected of conveying the infection should be examined, if they are available. Of all these points of differentiation the early and continued eosinophilia is perhaps the most useful.

**Prognosis.**—The prognosis depends on the severity of the infection, which varies much in different epidemics. The average mortality is low, between 1 and 2 per cent. Severe initial symptoms point to a heavy infection, but the accompanying vomiting and diarrhoea remove many worms, and so do not lead to a correspondingly severe second stage. In fact, persistent diarrhoea is to be regarded as of favourable import. Children very seldom die of the disease, and in them the prognosis is always more favourable than in adults. Death seldom occurs after the seventh week.

**Treatment.**—Treatment must be directed primarily to getting rid of as many adult trichinellæ as possible. It will be remembered that every adult produces about 1,500 embryos, and that they continue to be liberated for several weeks. The greater the number and the more rapidly the adults are removed, the less severe the later stages of infection. No anthelmintic is known which can be relied upon to kill the worms, though  $\beta$ -naphthol, thymol, and oil of eucalyptus are recommended by some. The best treatment is to give periodic doses of calomel during the first four or five weeks. The dose must be large (10-20 gr.), to ensure free purgation. If the patient is seen soon after eating meat which is found to be infected, an emetic should be given at once and calomel later. Once the embryos have reached the lymph-stream, no drug has any detrimental effect on their further development, and it is quite useless to give parasitocides for this purpose.

Treatment during the second stage must be

## TRIGEMINAL NERVE, PARALYSIS OF

adapted to ameliorate special symptoms as they arise, and to keep up the general condition, as in any other long-continued fever. For the pains, hot baths are beneficial. Acetylsalicylic acid (aspirin) may alleviate them, but it is often necessary to resort to morphia. For the profuse sweats, atropine in a pill (gr.  $\frac{1}{100}$ ) or some other preparation of the alkaloid is most effective.

**Prophylaxis.**—Preventive measures are the most useful. To feed pigs on raw pork and offal is dangerous. All products derived from the pig should be cooked very thoroughly. In Germany, where neither of these precautions is taken, pieces of muscle from all slaughtered pigs are examined microscopically, and infected carcasses condemned, but in Great Britain pigs are so rarely infected that this is unnecessary.

E. A. COCKAYNE.

**TRICHINOSIS** (see TRICHINELLIASIS).

**TRICHOPHYTOSIS** (see RINGWORM).

**TRICUSPID-VALVE DISEASE** (see VALVULAR DISEASE, CHRONIC).

## TRIGEMINAL NERVE, PARALYSIS OF.

—This nerve consists of a motor branch that supplies all the muscles of mastication, and a sensory portion that subserves sensibility on the anterior part of the scalp and the whole of the same side of the face. The motor root takes origin from a nucleus in the central grey matter at about the middle of the pons, while the sensory fibres, which spring from the cells of the Gasserian ganglion, terminate in a column of grey matter that extends from the middle of the pons to the second cervical segment of the cord. Owing to this long intramedullary course, pontine and bulbar lesions are very liable to produce sensory disturbances on the face. These occasionally occur also in syringomyelia. The nerve may be damaged by meningitis at its origin from the pons, and by inflammatory lesions and tumours as it lies upon or passes through the base of the skull. Sarcoma of the middle fossa is a not infrequent cause.

Disease of the sensory root or of its branches produces disturbances of sensation on the same side of the face, in a region varying with the fibres that are damaged. When the nerve is irritated by an inflammatory focus, by a compressing tumour or by a meningitis, the anæsthesia is usually preceded by sensations of numbness or of tingling, or by pain. It is

## TRISMUS

important to distinguish this pain from that of facial neuralgia. Its character is suggestive, as it is usually a continuous gnawing pain, while that of facial neuralgia is intermittent and sharp, stabbing or burning. Then neuralgia is not associated with anæsthesia or diminution of sensibility, nor with absence of the corneal reflex, which is the most delicate sign of involvement of these sensory fibres. The buccal and nasal mucous membranes become insensitve too, and smell and taste may be lost or blunted on the same side owing to the trophic changes that occur here. The most important complication is inflammation of the cornea and keratitis, which may lead to destruction of the eye or permanently impair its sight. It is now generally regarded as the direct result of anæsthesia of the eye which allows the cornea to be irritated by foreign bodies; it does not develop when the lids are sewn together or if the eye is kept covered. Herpes, due to inflammatory and other lesions of the Gasserian ganglion, is not uncommon; it rarely produces serious scarring, except on the cornea.

Lesions of the motor branch paralyse the masseter, temporal, and pterygoid muscles on the same side; mastication is consequently difficult, and when the mouth is opened the jaw deviates towards the affected side. Unilateral palsy does not inconvenience the patient seriously, but bilateral weakness may make it impossible for him to chew solid food.

Spasm of the masticatory muscles is dealt with in a separate article (see TRISMUS).

GORDON HOLMES.

**TRIGEMINAL NEURALGIA** (see NEURALGIA).

**TRIONAL POISONING** (see POISONS AND POISONING).

**TRISMUS** (Masticatory Spasm).—Most often trismus is an accompaniment of generalized convulsions. It is an early symptom in tetanus, though it may be preceded by spasm and rigidity in the neighbourhood of the infected wound. In tetanus neonatorum usually the first symptom noticed by the mother is the inability of the baby to take the breast. In strychnine poisoning the jaw muscles are, as a rule, affected later than those of the limbs. Trismus is met with sometimes in tetany and in hysteria. Dental caries and exposure to cold are occasional reflex causes, whilst it may be originated centrally by local disease

## TROPICAL ABSCESS

in the neighbourhood of the motor nucleus of the fifth nerve. Impaction of a "wisdom" tooth is a not uncommon cause. It is generally supposed to act by causing spasm reflexly, but is more often due to actual inflammatory invasion of the temporal muscle.

The teeth may be set close together or be separated by only a small interval, and the masticatory muscles stand out as firm bands. The spasm is often painful. Great difficulty may be experienced in administering food, which it may be necessary to give by a nasal tube. The treatment generally is that of the primary condition. FREDERICK LANGMEAD.

**TROCHLEAR PALSY** (see OPHTHALMOPLAGIA).

**TROPICAL ABSCESS.**—Suppurative hepatitis, produced by the *Amœba histolytica* (see DYSENTERY, and prone to lead to the formation of one or more large collections of pus, usually surrounded by dense fibrous walls.

**Geographical distribution.**—Although most frequently met with in tropical and sub-tropical climates, as in Southern Asia, Egypt, the Southern States of America, and all countries where amœbic dysentery is prevalent, it may develop in cold climates in subjects who have previously suffered from amœbic colitis.

**Etiology.**—The causation of the disease, particularly its relationship to dysentery, has been much disputed, but it is now established that it is always secondary to amœbic ulceration of the large bowel. Not infrequently, however, this is of a latent nature and has not caused any active dysenteric symptoms. I have found evidence, either clinical or post mortem, of former dysentery in 95 per cent. of liver abscesses. The amœbæ are constantly found in the walls of the abscess cavities, although they may be difficult to detect in pus removed by the aspirator.

The very great majority of the abscesses, including those in the earliest stages, are free from bacteria on first being opened.

Alcohol, even in moderate quantities, is a powerful predisposing cause of the disease.

Patients under 20 years of age are rarely attacked, most of the cases occurring in persons between 21 and 50. Women are remarkably immune to the disease; in only 2.7 per cent. of natives and 4.4 per cent. of European cases in Calcutta hospitals were the patients of this sex.

The disease is now common in natives in

## TROPICAL ABSCESS

India, although earlier writers found it to be rare, the modern increase probably being associated with the spread of alcoholic habits among the native races.

**Pathology.**—The amœbæ invade the tributaries of the portal vein in the submucous coat of the large intestine and, reaching the liver by that route, produce in it lesions varying widely in their nature. In acute cases of amœbic dysentery numerous abscesses, some very minute and containing only amœbæ, may be found, but these prove fatal before localizing symptoms, apart from a general acute hepatitis, become evident. In chronic or latent cases of amœbic colitis, one or more large abscesses may develop very insidiously and become surrounded by a well-defined fibrous wall, separating the abscess contents from surrounding healthy liver tissue and containing numerous amœbæ. In Calcutta, in a series of fatal cases, a single large abscess was found in half, two or more large ones in one-fourth, and multiple small abscesses in the remaining fourth. If those cases which recovered are taken into account, the abscess was solitary in 70 per cent. of the total number. Of single liver abscesses, one sixth are in the left lobe and the remainder wholly or partly in the right.

When a fibrous wall has developed, the cavity expands without further damaging the liver tissue, so that 3–6 pints of pus may be present without more than a small fraction of the organ being destroyed, and complete recovery may ensue under proper treatment. If not detected and suitably dealt with, the abscess may extend beyond the liver, most commonly through the diaphragm into the right lung, when it usually opens into a bronchus and discharges. The pericardium, pleural cavities, stomach, colon, and rarely the inferior vena cava or peritoneal cavity, may similarly be invaded. Supra- and perihepatic abscesses have also been described, but these are nearly always formed originally within the liver substance, the outer wall of the cavity having merged with surrounding structures. Left-lobe abscesses often point in the epigastrium, and may be mistaken for suppuration within the abdominal wall.

**Symptomatology.**—Although the onset is generally marked by fairly definite symptoms of hepatitis, it is very important to bear in mind that large collections of pus may form in the liver in a most insidious manner, with no local symptoms beyond slight enlargement

of the organ, and even with no fever when the patient comes under observation. Indeed, an actual or relative leucocytosis may be the only clue to the true nature of the illness. In more acute cases it is often exceedingly difficult to determine whether suppuration has or has not taken place. The decision is of great practical importance, as in the presupplicative stage the disease can be cured easily and quickly, and dangerous abscess-formation averted, whilst exploratory puncture may result, though very occasionally, in fatal internal hæmorrhage.

**The presupplicative stage.**—When the disease begins characteristically with the symptoms of acute hepatitis the liver is enlarged and very tender, and fever, usually of an intermittent but occasionally of a remittent nature, may persist for several weeks in the absence of actual suppuration. The duration of the symptoms in European patients, who come early under observation, was over one month before abscess-formation was detected in half the cases, whilst in 82 per cent. it was upwards of two weeks. Leucocytosis, often without pronounced increase of the proportion of the polynuclears, is a constant and useful sign at this stage. In a fair proportion of cases there are no definite symptoms referable to the liver, and only the discovery of leucocytosis in an obscure fever enables the condition to be recognized early enough for abscess-formation to be prevented.

**The suppurative stage.**—The symptoms which are present after actual abscess-formation has taken place do not differ from those of the presupplicative stage as long as the abscess remains within the substance of the organ, and has not attained so large a size that it causes definite localizing signs.

Until recently the only means of ascertaining if pus had collected in such cases was to perform an exploratory puncture—a procedure which is not always so harmless as it is often thought to be. The discovery of the easy cure of the presupplicative stage has largely removed this difficulty, for, in the absence of suppuration, the fever and the tenderness and enlargement of the liver rapidly subside under treatment, while the leucocytosis disappears. If the white corpuscles remain persistently high after the acute symptoms have disappeared, a localized or encysted collection of pus, requiring removal by the aspirator, is probably present.

**Local symptoms.**—Native patients often come under observation at a late stage when localizing symptoms may be quite evident; these

## TROPICAL ABSCESS

vary with the position of the abscess. The commonest site is in the upper part of the right lobe just beneath the diaphragm. Pain in the right shoulder is then a fairly constant symptom, and the liver dullness is increased, mainly in an upward direction. X-rays are of great use in this connexion, for by them the diaphragm is seen to be higher than normal and quite immovable on the right side, whilst a shadow may be evident at the base of the right lung, if the disease is spreading in that direction. Increased density of the liver shadow in the position of the abscess may also be evident, but this by itself is not a very reliable sign. It must also be remembered that these X-ray appearances may be equally well marked in amœbic hepatitis without actual suppuration, but in that case the specific treatment will rapidly resolve the condition, even after the lung is involved. When the lower part of the right lobe is affected the downward enlargement is very evident, while in left-lobe abscesses epigastric swelling, sometimes with fluctuation, reveals the nature of the disease. Liver abscesses opening into internal organs other than the lung are rarely seen in these days of comparatively early operation.

**Diagnosis.**—The principal points in the diagnosis have already been mentioned. In anæmic patients only a relative (over one white to 500 red cells) instead of an actual leucocytosis may be present. In the absence of the localizing symptoms described above, the failure of the specific treatment to clear up the symptoms, and a persistent leucocytosis may be the only reliable indications of pus-formation. Exploratory puncture may then be employed more safely to locate the pus, and arrangements should be made to deal suitably with the abscess at the same time.

**Prognosis.**—Amœbic liver abscess is now a preventable disease and he who allows it to develop through failure to recognize and treat adequately the early presuppurative stage, incurs a serious responsibility.

When an abscess has formed, the prognosis is serious, although it has been greatly improved by recent methods of treatment. The mortality was formerly about 60 per cent., and varied from over 70 per cent. in right-lobe abscesses opened through the ribs to 12 per cent. in small left-lobe abscesses pointing in the epigastrium. In abscesses opening through the lungs the death-rate was about 50 per cent., but such cases do remarkably well under emetine treatment. By aspira-

tion, together with treatment by ipecacuanha and emetine, the mortality has been lowered in a recent series of over 100 cases of all classes reported by Major E. O. Thurston, I.M.S., to 23 per cent. In the much less acute type met with in patients invalided to Europe the mortality is lower than in a series including very acute and frequently multiple abscesses in the tropics. If more than one large abscess be present the prognosis is very grave, and multiple small ones are nearly always fatal, but are seldom recognizable during life. Under emetine treatment, however, it is possible for numerous small amœbic abscesses to be completely recovered from: this occurred in a patient in whom they were seen during life in the course of an abdominal section, and from whom pus containing amœbæ but no bacteria was withdrawn by a syringe.

In hot damp climates the open operation is almost inevitably followed by secondary infection through the air, which greatly retards the healing process, if the patient survive the exhausting discharges. If sterility can be maintained by some form of syphon drainage, healing is far more rapid, and the prognosis improved.

During the presuppurative stage the disease, as the writer has shown, can be cut short by efficient treatment with the specific remedy against amœbic infection—ipecacuanha and its active principle, emetine.

Within the last few years the prevalence of amœbic liver abscess among Europeans in Calcutta and in the British Army in India has been reduced from an average of 98 per annum in the decade ending with 1907 to only 6 per annum in a recent year, and the disease is fast becoming rare wherever correct treatment is carried out in the early stages.

**Treatment.** At the present moment there is much difference of opinion regarding the treatment of amœbic abscess. Until the last decade the open operation had long been in vogue and had replaced aspiration. After having demonstrated that amœbic liver abscesses when first opened are almost invariably sterile as regards bacteria, but that secondary infection of the cavities practically always ensues within a few days in the damp germ-laden atmosphere of Bengal, and that weak quinine solutions rapidly kill the amœbæ, I was encouraged to try repeated aspiration and injection of sterile quinine solutions into the abscess cavities. The specific action of ipecacuanha in amœbic dysentery, and its rapid

## TROPICAL ABSCESS

curative effect in the presuppurative stage of amœbic hepatitis, led me also to give this drug in all cases of liver abscess after aspiration. This combined treatment has reduced the mortality of liver abscess to one-third to one-fourth of its former rate. Still better results have been obtained with emetine, and many surgeons in Calcutta and elsewhere have given up the open operation in favour of aspiration and emetine treatment, except in special cases such as small pointing epigastric abscesses. Whenever possible, therefore, aspiration, which may sometimes be done under local anæsthesia, should be performed, and as much pus as possible removed. A grain of emetine in sterile solution may be injected either into the abscess cavity or subcutaneously, and repeated daily. If pus reaccumulates, this plan may be repeated once or twice, as even if drainage has eventually to be resorted to, the cases do better for the preliminary aspirations. If doubt arises as to whether the pus is re-forming, a blood-count is of service, for if the leucocytosis persist, pus should again be sought for. Even a liver abscess pointing in the epigastrium may be successfully aspirated a little to one side of the softened area of the skin. If pus still reaccumulates after two or three aspirations and a full course of emetine injections which have extended over two or three weeks, the pus must be evacuated.

When this has been done, some form of siphon drainage best enables the cavity to be kept sterile. A rubber tube may be inserted through a small incision. It should fit closely into the opening, and be carried into a bottle under the bed containing some antiseptic. The incision may be avoided by using the flexible sheathed trocar, devised by the writer, to aspirate the abscess, as it permits of siphon drainage through the sheath without removing it. Manson has invented a large-sized trocar through which a drainage-tube is passed, which serves a similar purpose. Should the cavity become infected, in spite of all care, solutions of permanganate or iodine are best for irrigation.

After a liver abscess has been drained in one of the above ways or by the open operation, which many surgeons still prefer, the healing of the cavity is greatly accelerated by the administration of ipecacuanha by the mouth, or, better, of emetine hydrochloride hypodermically, the amœbæ rapidly disappearing from the pus under its influence. I have no

## TRYPANOSOMIASIS

doubt that it is the specific action of this drug in killing the amœbæ within the body tissues that has caused the aspiration method of treating tropical liver abscess to be revived with so much success.

LEONARD ROGERS.

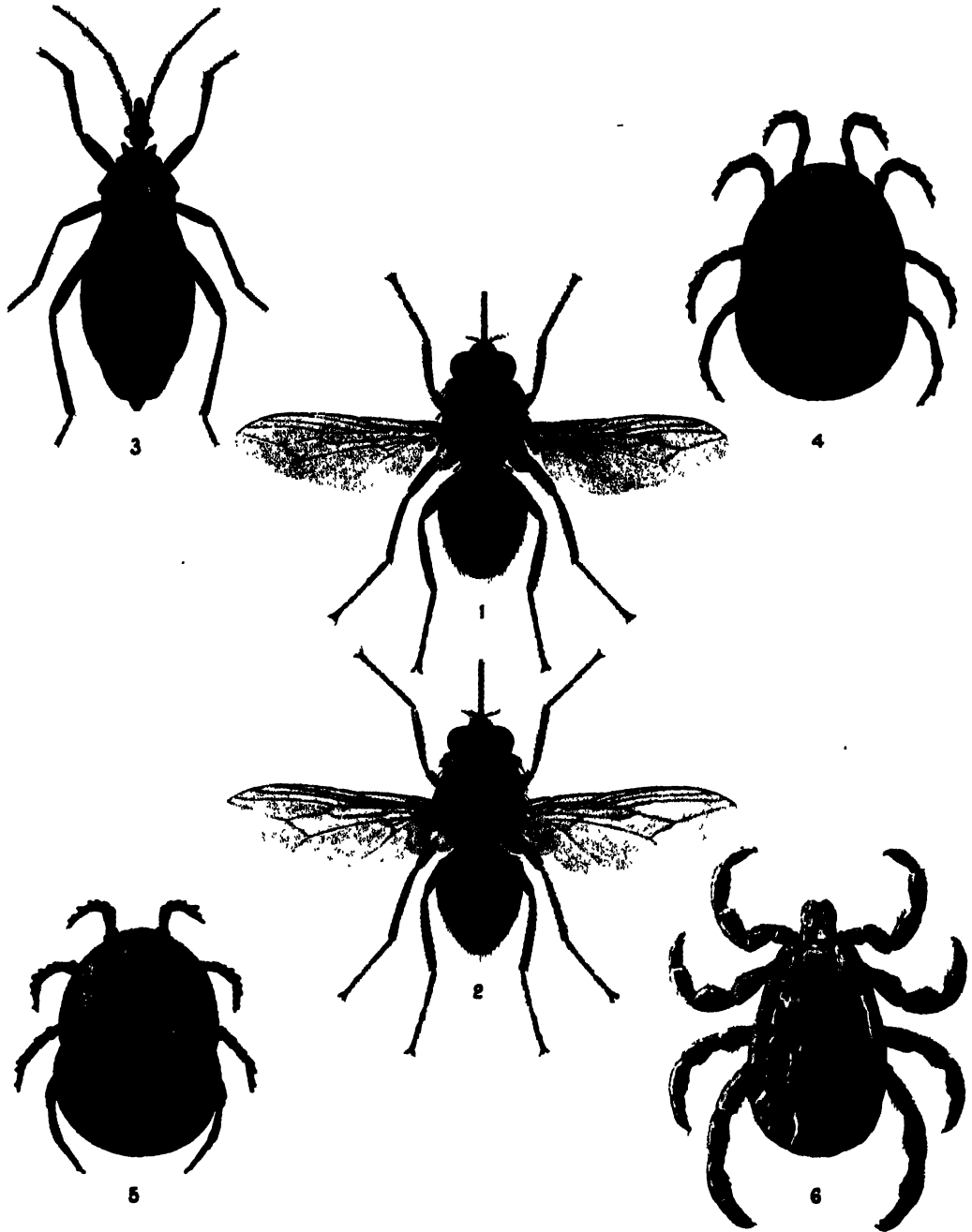
### TROPICAL SORE (see ORIENTAL SORE).

**TRYPANOSOMIASIS.**—An infection with a protozoan parasite of the genus *Trypanosoma*. There are at least three, and possibly more, forms of the disease, associated with as many different species of trypanosome.

**Geographical distribution.**—Trypanosomiasis is endemic in tropical and subtropical Africa. The earliest known reference to African trypanosomiasis, under the name of the "Sleeping Distemper," is by one John Atkins in "The Navy Surgeon" (1734). Chagas, in 1909, described another variety—South American trypanosomiasis—among the inhabitants of the State of Minas, in Brazil.

### AFRICAN TRYPANOSOMIASIS

Trypanosomiasis in Africa was probably at first confined to the west coast—Guinea Coast, Sierra Leone, Senegal, Gambia, St. Thomas, etc. With the advent and spread of European civilization and the consequent migrations of large numbers of natives, the disease extended across the continent, notably into the Congo State and to Uganda, where it was slaying a large percentage of the native population in the closing years of the last century and afterwards. At this time sleeping sickness was not known to have any connexion with trypanosomiasis, and the history of the discovery is briefly as follows. In 1901 Forde and Dutton discovered a trypanosome (*T. gambiense*) in the blood of a patient suffering from a peculiar fever on the Gambia. In 1902 Castellani found a trypanosome (*T. castellanii*) in the cerebro-spinal fluid of sleeping-sickness patients in Uganda. In 1903 Bruce and Nabarro amplified Castellani's observations and found trypanosomes in the blood and cerebro-spinal fluid of practically every case of sleeping sickness. They also established that the parasite was carried by a tsetse fly, *Glossina palpalis* (PLATE 38, Fig. 1), in which Kleine showed that *T. castellanii* undergoes a developmental cycle. Much work was done during the decade 1904–14, which confirmed and extended the discoveries of the above-mentioned investigators. It has further been shown that the sleeping sickness prevalent in Rhodesia is due to a different



1, *Glossina palpalis*, ♀,  $\times 3\frac{1}{2}$ . 2, *Glossina morsitans*, ♀,  $\times 3\frac{1}{2}$ . 3, *Conorhinus megistus*, ♀,  $\times 1\frac{1}{2}$ . 4, *Ornithodoros moubata*, ♀,  $\times 3$ . 5, *Ornithodoros savignyi*, ♀,  $\times 4$ . 6, *Dermacentor venustus*, ♂,  $\times 10$ .

PLATE 38.—PARASITE-BEARING TSETSE FLIES AND TICKS, ETC.



## TRYPANOSOMIASIS

trypanosome, *T. rhodesiense*, which is transmitted by *Glossina morsitans* (PLATE 38, Fig. 2).

Castellani summarizes the present distribution of African trypanosomiasis as extending "along the west coast of Africa from St. Louis, in Senegal, to Mossamedes, in Angola; from the coast to Timbuktu, on the Niger; through the whole of the Congo State into Uganda and Rhodesia; from Uganda and Busoga southwards to late German East Africa; and northwards into the Bahr-el-Ghazal Province of the Sudan." It must be remembered that the tsetse fly is probably an indispensable factor in the spread of the disease, and that patients going into a fly-free area do not act as potential originators of an epidemic.

**Etiology.**—There are probably at least

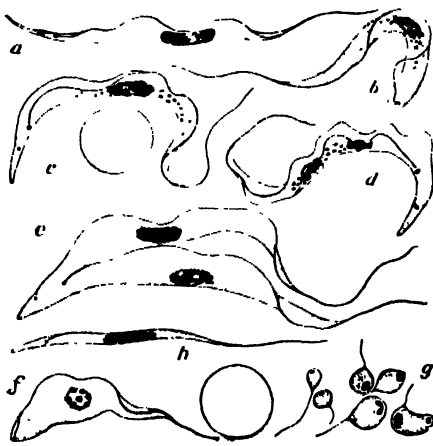


Fig. 97.—*Trypanosoma gambiense*: various forms from blood and cerebro-spinal fluid.

A, Elongated posterior extremity; b, blunt ditto; c, d, e, dividing forms; f, g, probably sexual forms; h, small round forms from cerebro-spinal fluid.

three types of African trypanosome pathogenic for man: *T. gambiense* (Fig. 97), spread by *Gl. palpalis* or *Gl. tachinoides*, and of which man is the principal vertebrate host; *T. castellanii*, spread by *Gl. palpalis*, and of which man is again the chief host; and *T. rhodesiense* (Fig. 98), spread by *Gl. morsitans*, and of which some believe the hartebeest and other animals are the vertebrate hosts. Other types (e.g. *T. vivax*), which are essentially animal trypanosomes, may occasionally infect man. When studying the identity and speci-

ficity of trypanosomes, a consideration of the morphological characteristics alone is not sufficient. It is necessary to study also the clinical picture of the disease in man, cross-immunization, and other animal experiments,

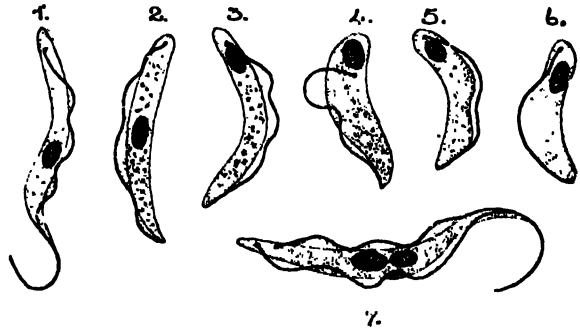


Fig. 98.—*Trypanosoma rhodesiense*. (After Laveran.)

1, 2, Normal forms in blood of man; 3-6, various stages of posterior displacement of the nucleus; 7, a dividing form

and the serum reactions. Studied in this way, *T. gambiense* is the least and *T. rhodesiense* the most virulent of the three African types.

**The parasite** (Fig. 99). A trypanosome is a worm-like body which in the living state is seen to be actively motile. It varies in length from 12 or 14  $\mu$  to 31 or 33  $\mu$ , short, medium, and long forms all occurring in the blood. Its width varies from 1.5 to 2.5  $\mu$ . The elongated cytoplasm is bordered by a wavy, fin-like undulating membrane. There are two nuclei—a large, oval trypomastix situated generally about the middle of the body, and a small kinetoplast situated usually near the anterior or flagellar end of the body. A filament of chromatin extends from the kinetoplast along the free border of the undulating membrane and thence along the attenuated posterior end of the protoplasmic body of the trypanosome, to end in a free flagellum of variable length. Often there is a vacuole just behind the kinetoplast, and there may be

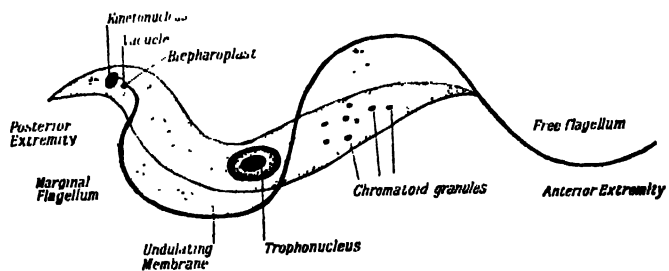


Fig. 99.—Schema of trypanosoma. (After Doell and Manson-Bahr.)



## TRYPANOSOMIASIS

chromatic granules in the cytoplasm behind the trophonucleus. Variations of the above type occur, such as short stumpy forms with no free flagellum, forms with the nucleus at or near the flagellar end, forms showing all stages of longitudinal fission, etc.

The life-history of the African trypanosomes in the human body is not accurately known, but "latent forms," consisting mainly of the two nuclei in a small amount of circular cytoplasm, probably occur in some of the internal organs. When trypanosome-containing blood is sucked by the appropriate tsetse fly, the fly remains *directly* infective for about twenty-four hours, but is non-infective for the next ten or twelve days, during which time the trypanosomes are undergoing development in its gut. After this a new generation of trypanosomes is produced in the salivary gland, thus rendering the fly infective when next it feeds on its vertebrate host. Fortunately only about 5-8 per cent. of the flies which feed on trypanosome-containing blood become infective. A somewhat similar but shorter cycle occurs in the bug which is the carrier of *T. cruzi*.

No race is immune, and age and sex have no influence as predisposing causes. Owing to the peculiar distribution of the fly, however, persons living or working along watercourses or the shores of lakes are liable to contract the disease.

### **Pathology and pathological anatomy.**

The trypanosome reaches the blood either directly or, more probably, via the lymphatic system, thereby inducing the glandular enlargement which is so constant a feature of the disease. This infection of the lymphatic system leads ultimately to changes in the central nervous system (meningo-myelo-encephalitis) with proliferation of the neuroglia and a perivascular cell infiltration. These two pathological processes, by compressing the small blood-vessels, lead to anæmia of the brain and spinal cord with consequently reduced metabolism in the nerve-cells. The result is the syndrome to which the name sleeping sickness is given. Secondary bacterial infections, especially with streptococci and pneumococci, frequently supervene and help to carry off the patient.

The cerebro-spinal fluid in the stage of trypanosome fever is practically normal, but in sleeping sickness it may show a slight turbidity, and increase of albumin and globulin. On centrifugalization there is usually a slight sediment which, on microscopic examination,

reveals a few lymphocytes and endothelial cells and trypanosomes in practically every case. In complicated cases streptococci or other organisms may be present.

Post mortem, the body is usually emaciated and the lymphatic glands are enlarged. The most obvious changes are seen in the central nervous system. The intracranial pressure is increased with flattening of the convolutions, and the sulci are filled with a turbid exudate having a "ground-glass" appearance. The pia arachnoid may be thickened and in places adherent to the grey matter, which is usually congested. Similar changes may be present in the spinal cord. The lymphatic glands are enlarged and inflamed, or in the later stages fibrotic, or they may show suppurative foci. The lungs may be pneumonic, the liver and spleen enlarged. Petechial subserous hæmorrhages are sometimes present.

Microscopically, the most characteristic change is the perivascular cell-infiltration first described by Mott. This is most marked, and appears earliest, where the cerebro-spinal fluid is most abundant, namely, around the vessels of the medulla, pons, cerebellum, and base of the brain. The first stage is a proliferation of the neuroglia elements, then a proliferation of the endothelial cell nuclei, and an infiltration of the pia arachnoid with lymphocytes which may become transformed into plasma cells.

Mott has also described somewhat similar changes in the lymphatic glands—increased vascularity with lymphocytes in all stages up to the formation of large plasma cells; degenerated, swollen plasma cells ("morula" cells), proliferated endothelial cells, hyperplasia of the trabeculae and walls of the lymph sinuses and vessels, leading eventually to fibrosis of the whole gland.

The internal organs may be hyperæmic, with sometimes a small round-celled infiltration.

**Symptomatology.**—Two distinct stages of the infection can be recognized—(1) a febrile or glandular stage—so-called trypanosome fever, and (2) a cerebral stage—so-called sleeping sickness.

The usual *incubation period* varies from two to three weeks; exceptionally it may be only ten days or may extend to months or even years. The bite of an infected tsetse fly may give rise to very little local irritation, so that a patient may not know exactly when the infection took place.

The symptoms of **trypanosome fever** are: (1) Recurrent pyrexia, the attacks lasting a week

## TRYPANOSOMIASIS

or more at a time and being of a remittent or intermittent type. (2) Fugitive erythematous eruptions in Europeans, especially on the trunk; in natives these eruptions are not observed, but the skin is often found to be dry and scaly; localized oedemas are present in some cases. (3) Increase in the pulse and respiration rates during the febrile attacks, with persistence of the pulse-rate afterwards. (4) Enlargement of the lymphatic glands, especially in the posterior triangle of the neck. (5) A general, deep hyperæsthesia.

This stage of the disease may be reckoned in weeks or may extend over several months or even years, and is curable. Many sufferers, however, die from intercurrent infections such as pneumonia, dysentery, etc., their general vitality and resistance being lowered by the trypanosome infection. Most of the survivors eventually pass into the second stage—namely, **sleeping sickness**, in which the central nervous system is invaded by the trypanosomes. Probably the varying course of the disease in different parts of Africa is correlated with the hypothesis that the trypanosomes (*gambiense*, *castellani*, and *rhodesiense*) are different species and not merely one species with varying degrees of virulence.

With the advent of the cerebral or sleeping-sickness stage the patient's habits and disposition change, a symptom which is often noticed by his friends and associates, whereas a stranger might not see that there was anything amiss. He becomes irritable or dull and apathetic, and careless about his work, dirty in his habits, and awkward in his movements. He does not sleep excessively as a rule—so that the common name "sleeping sickness" is a misnomer—but rather is apathetic, from which condition he can at first be easily aroused. Fine tremors of the tongue, arms, and hands are often an early symptom and become more marked as the disease progresses. The gait is at first shuffling, with possibly some inco-ordination. Later, the patient becomes bed-ridden with the knees bent and the thighs flexed on the abdomen. There is nothing abnormal to be noted about the special sense organs. Epileptiform seizures sometimes occur. During the earlier weeks of the illness there are usually pyrexial attacks—up to 102° or 103° F., the temperature falling each day to normal or below. Periods of apyrexia then occur, and during the last week or ten days of life the thermometer may register as low as 93° or 94° F. The pulse-rate may be increased apart

from any fever, and the pulse is usually small, quick, and of low tension. The facial expression is often dull and heavy—"mask-like"; the speech weak and indistinct. Constipation is pronounced in most cases, especially towards the end.

The lymphatic glands are usually enlarged, and trypanosomes may be detected in the gland juice obtained by puncture. An enlarged liver and spleen may possibly be due to a complicating malaria.

The blood picture in trypanosomiasis is usually complicated by coincident infections with other animal or bacterial parasites, but with this reservation we may say that trypanosomiasis causes a gradual anæmia affecting the red cells and hæmoglobin. Sometimes, however, the red cells may be increased to as many as 8,000,000 per c.mm. Auto-agglutination of the red cells is sometimes seen in fresh blood-films and may be of diagnostic value. The leucocyte count as a rule is not high, but the mononuclear cells are relatively increased in the earlier stages of the disease. With the advent of the secondary or terminal bacterial complications the polymorphonuclear cells increase in number. Trypanosomes are always scanty in the blood, and appear to show a certain periodicity. In the fever stage the parasites may be found in ordinary blood-films stained by Leishman's or some other modification of Romanowsky's stain. In the sleeping-sickness stage they are rarely found in blood-films, but can usually be observed in the sediment after centrifuging 10 c.c. of citrated blood three or four times.

In the last stages of the disease there is incontinence of urine. The patient by this time is quite lethargic and helpless, and takes food with the greatest difficulty, or has to be forcibly fed. The temperature is subnormal—92° to 95° F. or even lower. The pulse is almost imperceptible. Bed-sores may form owing to the incontinence of urine and sometimes of fæces. The patient becomes comatose, or there may be epileptiform seizures, and the final picture of the emaciated, flexed body of a patient dying of sleeping sickness is very like that of a general paralytic. The two diseases are indeed closely allied in their causation, symptomatology, and pathological anatomy and histology.

**Complications.**—Laryngitis, pneumonia, oedema of the lungs and glottis, secondary bacterial infections such as septic cerebro-spinal meningitis, suppuration of the lymphatic

## TRYPANOSOMIASIS

glands and bedsores may occur. It should be remembered, of course, that the patients may also be infected with other animal parasites, such as those of malaria, filariasis, ankylostomiasis.

**Duration.**—Trypanosome fever may last for several years, and with appropriate treatment a small percentage of the cases recover. Sleeping sickness itself is almost invariably fatal, this stage of trypanosomiasis lasting from a few weeks to six or eight months—rarely longer than a year. The variety of the disease produced by *T. rhodesiense* is usually more rapidly fatal than that produced by *T. castellanii*, and this in turn is more virulent than *T. gambiense*.

**Diagnosis.**—In the fever stage the disease is often diagnosed as malaria or other fever. The chief points in the diagnosis (apart from the finding of the parasite) are (1) absence of response to quinine, (2) tachycardia and rapid changes in the pulse-rate independent of changes in the temperature, (3) the erythematous eruptions—in Europeans, (4) the muscular hyperæsthesia, (5) the fine tremors of the tongue, and (6) the enlargement of the cervical lymphatic glands—though this may be due to other causes. Sleeping sickness may be confounded with general paralysis, tabes, syphilis, and tumour of the brain, beriberi, uræmia, and encephalitis lethargica. In *tabes* and *general paralysis* there is no hectic temperature as there is in trypanosomiasis; inco-ordination of movement is the dominant symptom in tabes, and in general paralysis the cerebral symptoms are as a rule more marked than in sleeping sickness. The Wassermann reaction is strongly positive in nearly all cases of general paralysis and in many cases of tabes, whereas it is probably negative in sleeping sickness uncomplicated by syphilis. Peripheral neuritis is the dominant symptom in *beriberi*, and the tremors, fever, and lethargy of sleeping sickness are absent. In *chronic uræmia* the urine is albuminous and œdema is usually a marked feature. *Encephalitis lethargica* runs a more rapid course than sleeping sickness, and there is often involvement of the cranial nerves.

The district from which the patient comes often furnishes a valuable clue to the diagnosis. In doubtful cases the blood, glands, or cerebro-spinal fluid should always be examined for the causal parasite. The following are the methods recommended: (1) Films of peripheral blood from ear or finger stained with Leishman—often negative. (2) Examination of stained blood-films from the erythematous eruption—

better than (1) but often negative. (3) Repeated centrifuging (three or four times) of 10 c.c. of citrated blood and direct examination of a drop of the third or fourth sediment. Actively motile trypanosomes can practically always be found by this method. (4) Puncture of an enlarged cervical gland and direct examination of the drop of fluid thus obtained. This method, too, is very valuable. (5) Examination of the sediment obtained after centrifuging 10 c.c. of the cerebro-spinal fluid. Actively motile trypanosomes are practically always found in sleeping sickness, but not in the fever stage. (6) If methods (1) to (5) fail to demonstrate the parasites, 10 c.c. of blood or spinal fluid may be injected into a susceptible animal—monkey (*Macacus rhesus* or *Cercopithecus ruber*), guinea-pig, dog, or rat.

**Treatment.**—Arsenic in some form is the only drug which until recently was found to be of any value in the treatment of trypanosomiasis. The most successful results have been obtained with atoxyl, which is sodium para-aminophenyl arseniate and contains about 25 per cent. of arsenic. After prolonged treatment with this drug the trypanosomes become atoxyl-resistant, and to prevent this phenomenon from occurring various forms of combined medication have been devised. Of these, the most valuable is probably the combined treatment with atoxyl and antimony, the latter as tartar emetic, sodium or potassium antimonyl tartrate, or orpiment. Atoxyl is injected intramuscularly, tartar emetic and the tartrates are injected intravenously, and orpiment is given in pills. Authorities differ as to the details of dosage of the respective drugs and the intervals between their administration. A small dose of atoxyl, 3 gr., can be injected every third day, or a larger dose, 5 or 7½ gr., every five days, or even 1 gm. at 10- to 20-day intervals. As much as 10 to 30 gm. of atoxyl may be given in a year, but toxic symptoms, especially those threatening blindness, must be guarded against. The dose of antimony is usually 5-10 cg., well diluted, intravenously. This can be repeated every few days, and a gramme or more may be given in the year.

A new compound of antimony—"stibenyli" (Allen & Hanburys)—has been tried, when atoxyl and tartar emetic can no longer be tolerated, with apparent promise of success. The intrathecal injection of the patient's serum obtained shortly after an intramuscular injection of atoxyl or an intravenous injection

## TRYPANOSOMIASIS

of stibenyl is worthy of trial when trypanosomes are actually present in the cerebro-spinal fluid. A patient cannot be regarded as cured until at least two injections of blood into susceptible animals fail to infect.

General treatment, such as hygiene, good food, and removal from the infected area, and symptomatic treatment should both be on ordinary lines.

**Prophylaxis** is a difficult and complicated problem, and must be considered under general and personal measures. Under the former come (1) segregation of the sick in districts free from tsetse fly; (2) control of the movements of non-infected natives; (3) the establishment of inspectorial stations to prevent infected persons from entering non-infected fly areas; and (4) clearing the bush near villages and along watercourses where the tsetse fly is usually found, more particularly at landing-places, fords, and ferries. For personal safety, protection from flies must be sought by wearing white clothes, high boots, and puttees or leggings. Fly-bites should at once be painted with iodine or formalin.

### SOUTH AMERICAN TRYPANOSOMIASIS

This form of trypanosomiasis is an acute or chronic disease occurring, usually among poor children, in Brazil, and is due to *Tr. cruzi* and spread by a bug. (PLATE 38, Fig. 3.) In the blood of a patient three forms of flagellated trypanosome occur, but when they enter the cells of the tissues, and more particularly those of the muscles of the back and limbs, they lose their flagella and resemble the Leishman-Donovan bodies. These multiply by binary fission and subsequently become flagellated again. During this process of multiplication the tissue cells dilate and eventually rupture, the parasites escaping into the tissues and setting up an inflammatory reaction. The muscular tissue, including the heart, is acutely inflamed; the spleen, kidneys, suprarenals and the lymphatic glands generally, may be inflamed and enlarged; the liver is enlarged and fatty; the serous membranes are also affected, with effusion into the various cavities; and the dura mater and pia arachnoid are congested or inflamed. The neuroglia cells, too, may be invaded by the parasites.

The acute form starts with a febrile attack—the temperature rising in the evening—and the trypanosomes may then be found in the blood. The thyroid, lymphatic glands, liver, and spleen are enlarged, and there is œdema

## TUBERCULIDES

of the face and other parts of the body. After repeated attacks the child may die, recover, or pass into the chronic stage.

Several varieties of the chronic stage are described: (1) a myxœdematous, with atrophy of the thyroid and the classical symptoms of myxœdema; (2) a pseudo-myxœdematous, with enlarged thyroid and a peculiar bronzing of the skin; (3) a cardiac, with various disturbances of the heart's action; (4) a nervous, with brain and spinal-cord symptoms, such as aphasia, bulbar palsy, and spastic paralysis.

The diagnosis is made by finding the trypanosomes in the blood during a febrile attack. The prognosis is grave, especially in the acute form. Treatment should be as for African trypanosomiasis. Prophylactically, the bite of the bug must be prevented. DAVID NABARRO.

**TSE-TSE FLY DISEASE** (*see* TRYPANOSOMIASIS).

**TUBAL ABORTION** (*see* PREGNANCY, EXTRA-UTERINE).

**TUBAL MOLE** (*see* PREGNANCY, MOLAR).

**TUBAL NEPHRITIS, CHRONIC** (*see* NEPHRITIS).

**TUBAL PREGNANCY** (*see* PREGNANCY, EXTRA-UTERINE).

**TUBERCULA DOLOROSA** (*see* NEUROMA).

**TUBERCULIDES.** —A group of affections of the skin and subcutaneous tissue, occurring in tuberculous subjects with a poor peripheral circulation, the lesions being symmetrical and, with one exception, lichen scrofulosus, affecting the extremities. The following types are recognized:

1. **Papular tuberculides lichen scrofulosus.** —The eruption consists of small papules about the size of a pin's head, of a yellow or dull-red colour, slightly raised above the surface. They are usually smooth, but may have a minute adherent scale. They occur in oval or rounded groups or rings on the sides of the trunk and in the lumbar region. They cause no symptoms and leave no scar. Lichen scrofulosus is most common in children suffering from tuberculous glands, bones, or joints, and I have seen it in association with extensive lupus of the face. An exactly similar eruption may follow the injection of tuberculin. Microscopical examination shows the lesions to have the characteristic

## TUBERCULIDES

structure of tubercles, and in exceptional cases Koch's bacillus has been demonstrated.

2. **Papulo-necrotic tuberculides.**—The eruption occurs in adolescents and young adults suffering from tuberculous glands, etc. The patients are always the subjects of bad peripheral circulation, chilblains, etc. The eruption occurs on the fingers, hands, forearms, elbows, feet, knees, ears, and in rare instances on other parts. The earliest stage is a small, hard, pinkish nodule in the skin. The colour deepens, and then a small pustule appears at the apex of the nodule. This may dry up to a crust, which falls after a few weeks, exposing a conical ulcer that slowly heals, leaving a depressed, pale scar. The lesions are rarely larger than a small pea; they come out in crops, especially in cold damp seasons, sometimes for several years. Various names are given to these eruptions, depending upon conditions that probably are purely accidental. The name *folliculis* has been given to those in which the lesions are believed to involve the follicles of the skin. *Acne cachecticorum* is another synonym.

The papulo-necrotic tuberculides have to be distinguished from chilblains and from lupus erythematosus, with both of which conditions they may be associated. The tendency to form conical ulcers leaving scars is the special point of distinction.

3. **Erythema induratum (Bazin's disease).**—This tuberculide of the subcutaneous tissue occurs chiefly in young girls in the form of red or purplish indurated swellings on the legs, particularly on the lower half, rarely on the thighs and upper extremities. These indurated swellings not infrequently break down into large, deep, irregular ulcers, which run a chronic course, if the patient is obliged to stand for long periods at her occupation. The lesions occur on both legs, as a rule, and their development thus more or less symmetrically in young subjects is a point of distinction from the tertiary syphilitic ulcer. In a doubtful case the Wassermann test should be applied. (*See also ERYTHEMA.*)

4. **Erythema nodosum.**—It is held by numerous authorities that erythema nodosum is a tuberculide, and in this connexion I may mention that I have seen cases of this type following the injection of tuberculin (bacillary emulsion). It is considered in the article on ERYTHEMA.

5. **A rare form of tuberculide** is an indolent type of neoplasm with no tendency to ulcera-

## TUBERCULOSIS, ACUTE GENERAL

tion, described by Darier and Roussy as *hypodermic sarcoid*. It occurs most frequently in women between 30 and 40 years of age.

**Treatment of the tuberculides.**—General treatment is all-important. The patient should be well fed, and removed if possible from bad hygienic surroundings. Iron and arsenic with cod-liver oil should be administered. I have seen benefit in some cases of the papulo-necrotic type, and also in Bazin's disease, from the injection of minute doses of tuberculin, beginning with  $\frac{1}{10000}$  mg. Darier recommends injections of the soluble salts of mercury or of calomel. In Bazin's disease the ulcers usually heal rapidly if the patient can be kept for a few weeks in the horizontal position. They should be dressed with the red oxide of mercury ointment.

J. H. SEQUEIRA.

## TUBERCULIN IN TREATMENT (*see* PULMONARY TUBERCULOSIS).

## TUBERCULIN TESTS (*see* SEROLOGICAL DIAGNOSIS).

**TUBERCULOSIS, ACUTE GENERAL** (*syn.* Miliary Tuberculosis).—Under this term are included forms of tuberculosis in which large numbers of tubercle bacilli escape into the blood-stream and thus become distributed widely throughout the body, causing the formation of miliary tubercles in lungs, meninges, spleen, liver, kidneys, serous membranes, and other structures. In nearly every case their source is readily discerned after death and is found to be an actively disintegrating tuberculous focus. The primary focus most commonly met with is a softening and caseous gland, intrathoracic or less often mesenteric; but it may be situated within the lung, the tonsils, or elsewhere. Otitis media, tuberculous arthritis, and spinal caries occasionally are sources. In children, in whom the disease is chiefly encountered, the original focus is glandular in approximately 90 per cent. of the cases. Careful dissection will usually demonstrate the invasion of a neighbouring blood-vessel, which is most often a pulmonary vein, but may be the superior vena cava or one of its main tributaries, a cerebral sinus, the aorta, or a peripheral vessel, according to the position of the causative focus. In one group of the cases the generalization commences by means of the thoracic duct.

Since the intrathoracic glands are so frequently the site of the primary disease and children are particularly affected, it is not

## TUBERCULOSIS, ACUTE GENERAL

surprising to find that diseases with respiratory complications, such as measles and whooping-cough, are frequently antecedent, either proximately or with an intervening period during which return to health is rarely complete. In adults, visceral tuberculosis, notably phthisis, provides the primary focus relatively more frequently. The escape of the bacilli into the blood-stream in sufficient numbers is determined in some cases by traumatism, a history of accident before the onset of symptoms being sometimes obtainable, whilst in others operative measures directed to tuberculous glands or joints constitute the immediate cause. The disease is invariably fatal.

**Clinical varieties.**—Though many cases do not conform to any group, certain fairly distinctive clinical varieties can be recognized. Such are the meningeal form, the general or typhoid form, the pulmonary form, and the glandular form, according to the site of the closest aggregation of miliary tubercles.

1. The first of these constitutes **tuberculous meningitis**, for which see Tuberculous Meningitis, under MENINGITIS.

2. The **general or typhoid form**, as its name implies, bears some clinical resemblance to typhoid fever. There is often a period of general malaise and ill-health before the acute symptoms develop, though sometimes the onset is sudden and the course rapid from the beginning. Headache and torpor accompany the fever, which is generally irregular, but may be remittent, intermittent, or inverted, as in pulmonary tuberculosis. In other cases there may be no rise or only a transient rise of temperature. The toxæmia becomes profound, sometimes so quickly as to suggest the escape not only of bacilli but also of a dose of their toxins into the blood-stream. The pulse becomes quick, the respirations are increased, and slight cyanosis may develop. Sweating is prominent and wasting rapid. There is often a cough, while examination of the lungs reveals slight bronchitis with perhaps crepitations, chiefly at the pulmonary bases. The abdomen may be somewhat protuberant and the spleen enlarged. Constipation is the rule, though diarrhoea may occur. With deepening torpor the countenance becomes of a dusky grey colour, Cheyne-Stokes breathing is often seen, and the patient dies in coma. Edema of the extremities or purpura may be noticed before death, while towards the end meningeal symptoms may appear.

From *typhoid fever* the disease is distinguished

by the absence of the characteristic rash, by failure to grow the *B. typhosus* from the blood, a negative Widal reaction, and an absence of the "staircase" rise of temperature. Sputum is rarely procurable, but, if obtained, may decide the diagnosis. The spleen is somewhat firmer than in typhoid fever, though this distinction fails in the acutest cases. The relatively slow pulse of typhoid fever is a valuable guide. The temperature is more erratic and the wasting more rapid than in typhoid. Ophthalmoscopic examination should not be omitted, for choroidal tubercles may provide the only diagnostic sign. Tubercle bacilli are seldom obtainable from the blood. Little faith can be placed either in a blood-count or in tuberculin reactions in these acute cases.

3. In the **pulmonary form** the same general symptoms of toxæmia are met with but are accompanied by features indicating respiratory disease. Cough and dyspnoea are more in evidence, while the degree of anoxæmia may be severe, leading to extreme cyanosis and jactitation. In many cases nothing can be detected clinically in the lungs except fine and medium-sized râles, first noticeable at the bases. Later, patches of consolidation from broncho-pneumonia and areas of collapse and emphysema may provide their particular signs.

The diagnosis is generally less difficult than in the former variety. A primary focus of tubercle has more frequently been recognized, and the rapid wasting, with pyrexia, intense cyanosis, and rapid downward course in the absence of definite pulmonary signs, suggests the explanation. *Miliary broncho-pneumonia* provides a difficulty which, in the absence of sputum, may be insurmountable, but the cyanosis is seldom so intense and the spleen is not enlarged. Choroidal tubercles should be looked for. Especially confusing is the diffuse broncho-pneumonia without signs of pulmonary consolidation sometimes met with in influenza, in which the cyanosis is also extreme and the spleen may be enlarged. The advent of signs of basal meningitis, such as a squint, with or without examination of the cerebro-spinal fluid may make the diagnosis clear.

**Treatment.**—There is no method of cure and no treatment is of value, except that directed to symptoms and to relief of distress during the period of consciousness.

4. The **acute glandular form** is described under LYMPHATIC GLANDS, TUBERCULOSIS OF.  
FREDERICK LANGMEAD.

## TUBERCULOSIS FROM THE STANDPOINT OF PREVENTIVE MEDICINE

### TUBERCULOSIS DISPENSARY (*see* TUBERCULOSIS FROM THE STANDPOINT OF PREVENTIVE MEDICINE).

#### TUBERCULOSIS FROM THE STANDPOINT OF PREVENTIVE MEDICINE.

—The present-day approach to tuberculosis stands in striking contrast to that of forty years ago. In 1882 the conception was recast through the discovery of the essential cause of the disease. The determination of the tubercle bacillus led to precision of thought and a gradual shifting of the outlook on tuberculosis.

**Historical.**—Prior to 1882, various observers—notably Klencke, Villemin, Cohnheim, and Klebs—had voiced the opinion that tuberculosis was an infective disease. The effect of their work on ordinary practice was, however, slight. Even the convincing demonstration of the tubercle bacillus by Koch required time to prove its practical value. None the less, there was thereby rendered available for the first time a sure criterion of what should be considered tuberculous. The detection of the tubercle bacillus made the diagnosis in many cases easy. The study of its life-history cleared up the pathology, and afforded scientific grounds for a rational scheme of prevention and treatment.

Up to that time the care of the tuberculous patient had been empirical. The misconception of the cause of the disease and the conditions which favour it, and the prognostic hopelessness gave rise to apathy in treatment and a fatal negation of effort. The picture of the consumptive in those days, waiting his turn at an out-patient department, was pathetic in the extreme—prolonged detention in a close room, hasty application of a stethoscope, perhaps the demonstration of physical signs to a group of students, and then the return to his unhealthy environment—his sole consolation, a prescription for some favoured cough mixture or a bottle of cod-liver oil.

The recognition of tuberculosis as essentially an infective process, whose spread is largely dependent on environmental conditions, has (a) progressively altered the attitude of the practitioner to the individual patient, and (b) widened the horizon of preventive medicine.

(a) The individual patient affected with tuberculosis—in lung or genito-urinary tract or elsewhere—is no longer regarded as suffering from a local lesion, inflammatory or ulcerative, of the given organ, but as the subject of a specific infection which, while manifesting

itself with more or less prominence in a particular organ, exerts its toxic influence throughout the system. It matters not by what channel infection has been contracted, the essential facts and the course of the process are the same. The recognition of this has made possible the treatment of tuberculosis on scientific lines.

(b) The determination of the infective nature of tuberculosis and its widespread, practically universal, distribution throughout the civilized world, has led to its inclusion in the group of diseases communicable from man to man and from animal to man. The result has been the institution of special measures in the interest of the community as well as of the individual.

The extraordinary change which has been effected in the course of forty years appears all the more striking when we recall that tuberculosis, under one or another name, had been known and studied from the earliest times. The portraiture of the phthisical patient drawn by Hippocrates bears the impress of a master in clinical observation. But the classic description depicts only the *terminal* effects of the disease. With Hippocrates, phthisis heads the list of diseases which are “inevitably fatal.” The finer details—the shading of health into disease, referable to the gradual advance of infection—escaped notice.

**Experimental method.**—Now that the morbid factor, the bacillus, has been isolated and the disease reproduced experimentally, the most effective key to the natural history of tuberculosis is the study of the spread of the disease in the infected animal from the primary seat of inoculation throughout the various tissues and organs. This is most readily done in the guinea-pig. The progressive dissemination of infection from the site of inoculation to successive glands and groups of glands, and thereafter to the several viscera, may be followed in the gradually widening distribution of tuberculous deposits and associated advance of systemic intoxication, finally culminating in death.

**The inoculated guinea-pig.**—The effects produced by inoculation with the bacillus depend on (a) the amount of the dose, (b) its repetition, (c) its virulence, (d) the resistance offered.

In every case of successful inoculation two sets of effects are traceable—(1) local, i.e. at the point of inoculation, and therefrom successively in glands, viscera, etc., and (2) general or systemic.

## TUBERCULOSIS FROM THE STANDPOINT OF PREVENTIVE MEDICINE

(1) *Local effects.*—Where a subcutaneous dose has been sufficiently large, there occurs, in the course of five to ten days, induration at the point of inoculation. When this suppurates and bursts externally, a sore or chancre is produced. Thereafter—from about the tenth day onwards—lymphatic glands in the immediate neighbourhood on the same side become infiltrated, and presently more distant glands, at first on the same side of the body, and then more widely. In the course of three weeks or so the deeper glands are found involved, and also the spleen, which becomes studded with tuberculous deposit. Within four weeks the involvement of the glandular system is widespread, including mesenteric and other abdominal glands, deep cervical, and bronchial. About the same date, the lungs are commonly found invaded. In the course of five to six weeks tubercle may be found in the kidney.

(2) *Systemic effects.*—With the extension of local lesions, general or systemic disturbance occurs, appetite tends to be reduced, and sooner or later the animal loses weight. Its fur becomes shabby, its movements are less active, and death usually occurs in from six to twelve weeks.

These phenomena supervene with remarkable regularity, the progress of events varying somewhat according to the dose. With an infinitesimal single dose no effects may be traceable. The validity of negative results is necessarily open to question, and the possibility of fallacies must be kept in mind. For the most part the result is positive.

**Environmental influence.**—The bearing of environment on the effect of inoculation is illustrated in certain observations of Trudeau in the rabbit. Fifteen healthy rabbits were divided into three groups, each containing five. The first group, after inoculation, was placed in the best surroundings and allowed to run wild in the fresh air and sunshine, with an abundant provision of food. The second group, similarly inoculated at the same date, was placed under bad conditions of environment, being kept in a small box in damp, dark surroundings with foul air and insufficient food. The third group was placed under similarly bad conditions but was not inoculated.

All the rabbits in the first group, with one exception, resisted inoculation or, if infected, recovered. Of the second group, all the rabbits but one died within three months, and in each

case the organs showed extensive tuberculosis. The several members of the third group were killed after an interval and, although emaciated, in no single instance showed a trace of tuberculosis.

**Inoculation of the human subject.**—Nature's methods of inoculation are slightly different from those of the laboratory. There is seldom the *abrupt* introduction, either subcutaneously or intraperitoneally, of a considerable quantity of bacilli. The dosage may be infinitesimal and the point of inoculation merely a devitalized mucous surface. On the other hand, the dose tends to be repeated from time to time, and this, for the most part, in a favouring environment.

The results in the laboratory animal and the human subject alike enforce the significance of (a) infection—without tubercle bacilli there is no tuberculosis; and (b) environment—in proportion as the immediate surroundings are physiologically good, more or less successful resistance to inoculation may be maintained. Unfortunately, in the human subject, where through the constant presence of a source of infection, e.g. in the household, the opportunity for inoculation is continued, the value of physiological environment is lessened. Further, for the most part, continued exposure to infection is associated with continuance of unphysiological environment.

1. *Local effects.*—Tuberculosis is induced like syphilis through the entrance into the system of the specific organism. The point of entrance may be theoretically anywhere—either a cutaneous or, much more commonly, a mucous surface.

For our present purpose we may confine attention to the mucous surface. Although tubercle bacilli may obtain a successful footing at any point, in the alimentary tract from the lips to the anal orifice, and in the respiratory tract from the nostril to the ultimate alveolus, certain situations are more likely than others. The great vulnerability of the tonsillar region, including the posterior nares and the fauces, must especially be kept in view.

Wherever inoculation takes place, whether about the fauces or in the intestine, or in the larynx or bronchus, the ultimate result is similar. Successful inoculation may be accompanied by little or no local change at the point of entrance. The mucous membrane may appear intact to naked-eye and even to microscopic examination. This is especially



## TUBERCULOSIS FROM THE STANDPOINT OF PREVENTIVE MEDICINE

likely if infection has taken place, as it chiefly does, during the tender period of developing childhood. At that date the mucous surfaces are more absorbent and succulent. To this is attributable the greater frequency of infection in childhood and, likewise, the infrequent evidence of primary lesion.

In common with syphilis, tuberculosis has a definite, if seldom traceable, point of inoculation. It differs from syphilis in the fact that primary local disturbance is slighter and more transient. It is noteworthy that when inoculation is cutaneous, as opposed to mucous, the local manifestations are more evident and lasting, recalling the features of the initial tuberculous sore in the guinea-pig, or the primary chancre of syphilis.

Wherever inoculation has occurred, whether through mucous membrane or skin, the further course consists for the most part in a *gradual spread by way of the lymphatic system*. This cannot be emphasized too strongly. First, one or two glands in the neighbourhood of the point of inoculation become enlarged, and then, progressively, others at a greater distance, and so on until the lymphatic system is widely involved. The extent of the spread depends on the degree of resistance which the patient offers. It may be, as is suggested by some observations, that the glands exert an antagonistic influence.

The enlargement of glands is not chiefly an enlargement obvious on the surface, characterized by gross deformity with associated caseation and perhaps softening, but consists in progressive infiltration in chain after chain of lymphatic glands, so slight in degree that it is for the most part missed. *If the detection of tuberculosis, especially in children, is to become really effective, the child's glandular system must be most carefully investigated.* Persistent enlargement of glands, in the cervical or axillary or abdominal or thoracic region, should be regarded as clinical phenomena, which call for definite explanation.

The further course differs according as the spread takes place by continuity of tissue, or by the lymph-stream, or by the blood-current. Sooner or later other structures, other viscera—bone, lung, kidney, or brain—become involved, with recognizable anatomical changes. These anatomical changes are associated with symptoms referable to the organ affected, and in many cases with determinable physical signs. Whether viewed pathologically or

clinically they constitute the *local* effects of invasion.

**2. Systemic effects.**—The local effects, important as they are, do not constitute the chief or ultimate danger of tuberculosis. The significant factor is the *systemic* intoxication which, sooner or later, results from the bacillary invasion. To this intoxication are chiefly referable the more important clinical effects and the fatal termination.

The *tuberculous toxins* seem to act especially on neuro-muscular structures. Their dystrophic influence is evidenced by the progressive loss of sarcous substance and associated irritability of muscle. To this are referable the early manifestations of motor weakness both of limbs and of viscera—the feeling of tiredness, disinclination for effort, softening pulse, and gastrointestinal sluggishness. These neuro-muscular effects are generally in evidence, even in pulmonary tuberculosis, long before cough or expectoration or other indication of local lesion is forthcoming.

**Prevalence of tuberculosis.**—There is much more tuberculosis in the world than is commonly supposed. The usual statement that from one-seventh to one-tenth of the civilized population die from tuberculosis is misleading.

The observations of Nägeli (1900), which showed that definite signs of tuberculosis were found in 97 per cent. of bodies examined consecutively by him, that is, of persons dying in a general hospital from all sorts of diseases and accidents, have not escaped critical comment. By many they have been regarded as exaggerated. Yet similar observations have been made by other workers. In October, 1917, they were strikingly confirmed by Reinhart (Berne), who, in a long series of consecutive post-mortem examinations, found that 96 per cent. yielded evidence of tuberculosis. The latter observer has carefully analysed the 96 per cent., dividing them into cases which showed present activity of lesion, and cases which showed more or less completely healed lesion. Thus, 32 per cent. were found to present evidence of progressive tuberculosis—that is, one-third of all the patients who terminated their days within the general hospital—while in the remaining 64 per cent. there was found evidence of healed tuberculosis.

**Mortality from tuberculosis in Great Britain and Ireland.**—The following tables record the number of deaths officially accepted as having occurred, in 1920, from the more frequent forms of tuberculosis:—

# TUBERCULOSIS FROM THE STANDPOINT OF PREVENTIVE MEDICINE

TABLE I

TOTAL DEATHS, AND DEATH-RATE PER 100,000 POPULATION, FROM  
TUBERCULOSIS, 1920

ENGLAND			SCOTLAND		
	<i>Total number of deaths*</i>	<i>Death-rate per 100,000 population</i>		<i>Total number of deaths</i>	<i>Death-rate per 100,000 population</i>
Tuberculosis (all forms)	42,545	113	Tuberculosis (all forms)	6,042	124
Pulmonary Tuberculosis	33,469	89	Pulmonary Tuberculosis	4,194	86
Tuberculous Meningitis	3,585	10	Tuberculous Meningitis	590	12
Abdominal Tuberculosis	2,192	5	Abdominal Tuberculosis	525	11
			IRELAND		
				<i>Total number of deaths</i>	<i>Death-rate per 100,000 population</i>
* These totals include the following deaths of non-civilians:			Tuberculosis (all forms)	7,651	171
Tuberculosis (all forms)	240		Pulmonary Tuberculosis	5,911	132
Pulmonary Tuberculosis	201		Tuberculous Meningitis	487	11
Tuberculous Meningitis	5		Abdominal Tuberculosis	481	11
Abdominal Tuberculosis	7				

TABLE II

LARGER CITIES OF GREAT BRITAIN AND IRELAND, 1920: DEATH-RATE PER 100,000  
POPULATION FROM TUBERCULOSIS

(In English towns the rates refer to civilian population only.)

	<i>Estimated population</i>	<i>Death-rate per 100,000 from</i>			
		<i>All Tuberculosis</i>	<i>Pulmonary Tuberculosis</i>	<i>Tuberculous Meningitis</i>	<i>Abdominal and other Tuberculosis</i>
London . . . . .	4,531,971	127	106	10	11
Glasgow . . . . .	1,098,568	149	106	15	27
Birmingham . . . . .	895,915	113	95	6	12
Liverpool . . . . .	803,452	173	141	13	19
Manchester . . . . .	770,597	143	117	10	16
Sheffield . . . . .	492,570	118	93	10	15
Leeds . . . . .	448,913*	158	125	14	19
Dublin area . . . . .	415,000	238	178	—	60
Belfast County Borough . . . . .	413,000	237	185	—	52
Bristol . . . . .	375,641	122	99	10	13
Edinburgh . . . . .	334,942	124	85	11	28
Bradford . . . . .	293,979	111	92	7	12
Newcastle-on-Tyne . . . . .	286,061	175	133	18	24
Dundee . . . . .	184,084	137	99	11	26
Aberdeen . . . . .	164,907	129	93	11	25

\* The County Borough of Leeds was extended on April 1st, 1920, and the population shown in this table is an adjusted population, i.e. weighted so as to be comparable with the deaths as registered throughout the year.

# TUBERCULOSIS FROM THE STANDPOINT OF PREVENTIVE MEDICINE

## TABLE III

DEATH-RATE PER 100,000 OF POPULATION, MALE AND FEMALE, IN SCOTLAND, AND COLLECTIVELY IN BURGHES AND DISTRICTS THEREOF, 1920, FROM VARIOUS FORMS OF TUBERCULOSIS

	<i>Estimated population</i>	<i>Death-rate per 100,000 from</i>				
		<i>All Tuberculosis</i>	<i>Pulmonary Tuberculosis</i>	<i>Tuberculous Meningitis</i>	<i>Abdominal Tuberculosis</i>	<i>Other Tuberculosis</i>
SCOTLAND . . .	4,864,396	124	86	12	11	15
Males . . .	2,317,538	134	92	13	10	18
Females . . .	2,546,858	116	81	11	11	13
Larger Burghs . . .	2,385,331	141	98	14	12	17
Smaller Burghs . . .	960,671	115	78	11	10	16
County Districts . . .	1,518,394	104	73	10	10	12

## TABLE IV

DEATH-RATE PER 100,000 OF POPULATION IN EACH AGE-GROUP IN ENGLAND AND WALES IN 1920 FROM PULMONARY TUBERCULOSIS, TUBERCULOUS MENINGITIS, ABDOMINAL TUBERCULOSIS, AND OTHER TUBERCULOUS DISEASES

	<i>Age</i>									
	<i>All ages</i>	-1	1	5	10-	15-	45-	55-	65-	75 and over
Pulmonary Tuberculosis . . .	89	25	22	12	26	120	135	107	73	31
Tuberculous Meningitis . . .	10	74	49	18	11	3	1	1	—	—
Abdominal Tuberculosis . . .	6	43	22	6	5	4	3	2	2	1
Other Tuberculous Diseases . . .	8	21	13	7	7	8	8	10	13	12

## TABLE V

DEATH-RATE PER 100,000 OF POPULATION IN EACH AGE-GROUP IN SCOTLAND IN 1920 FROM PULMONARY TUBERCULOSIS, TUBERCULOUS MENINGITIS, ABDOMINAL TUBERCULOSIS, AND OTHER TUBERCULOUS DISEASES

	<i>Age</i>											
	<i>All ages</i>	-1	1	5	10-	15-	25-	35-	45-	55-	65-	75 and over
Pulmonary Tuberculosis . . .	86	16	18	14	28	113	129	133	138	102	62	32
Tuberculous Meningitis . . .	12	132	50	19	12	5	2	1	1	—	—	—
Abdominal Tuberculosis . . .	11	61	34	14	10	8	5	4	5	6	3	1
Other Tuberculous Diseases . . .	15	46	21	12	12	16	12	12	13	18	11	16

# TUBERCULOSIS FROM THE STANDPOINT OF PREVENTIVE MEDICINE

## TABLE VI

DEATH-RATE PER 100,000 OF POPULATION IN EACH AGE-GROUP IN IRELAND IN 1920 FROM PULMONARY TUBERCULOSIS, TUBERCULOUS MENINGITIS, ABDOMINAL TUBERCULOSIS, AND OTHER TUBERCULOUS DISEASES

	Age											
	All ages	*-1	1-	5-	10	15	25-	35-	45	55-	65	75 and over
Pulmonary Tuberculosis . . .	132	*23	26	26	61	200	215	197	168	114	45	19
Tuberculous Meningitis . . .	11	*55	32	22	21	10	5	3				
Abdominal Tuberculosis . . .	11	*40	33	13	9	9	11	7	6	5	2	1
Other Tuberculous Diseases . .	17	*41	23	8	15	21	15	16	21	20	10	10

\* The rates for deaths under one year are per 100,000 births registered.

**Misleading statistics.**—In judging of the significance of these tables it must be kept in mind that a considerable number of persons die from tuberculosis, and yet the official return—from one cause or another—is given in terms of bronchitis, pneumonia, etc. This, which is far from a negligible error, was doubtless commoner in the past than it is now. Still, it must be admitted that the official figures represent quite insufficiently the actual number of deaths from tuberculosis.

Beyond this, it must be kept in mind that a considerable number of deaths justly recorded as *immediately* resulting from other causes such as acute illnesses, are none the less *indirectly* attributable to tuberculosis, which, in the given individual, was the primary devitalizing cause that accentuated the gravity of the acute illness (*cf.* the high mortality of measles).

**Declining death-rate.**—Fortunately, the mortality records, if still appalling, show a progressively favourable tendency. This is brought out by a comparison of the Registrar General's figures for 1890 and 1920—an interval of thirty years. From pulmonary tuberculosis alone, in 1890, the death-rate in England was 168 per 100,000, whilst in 1920 it was 89 per 100,000. From the same cause, in Scotland in 1890 the death-rate was 193 per 100,000, whilst in 1920 it was 86 per 100,000.

The comparison shows that by some means or other a successful resistance is now being offered to tuberculous invasion which was not offered before.

This is the more significant as it may be safely assumed that, with increasing exacti-

tude of diagnosis, the actual proportion of deaths properly registered has increased.

**Accelerating decline in death-rate.**—The progressive decline is noteworthy. A hasty glance at the figures might suggest that the decline has been continuous and equal from decade to decade. A fuller examination shows that this is not so. The fall has latterly been considerably accelerated.

Thus, in Scotland, taking twenty years up to 1890, the *percentage fall* in mortality from all forms was 35, while during the twenty years from 1900–1919 the *percentage fall* was 45.

In addition to the accelerating fall in the death-rate, it is noteworthy that the age at death is gradually being postponed.

That the remarkable fall is not due to natural causes which influence all civilized countries in more or less similar fashion is seen by comparing the figures for England and Scotland with the corresponding figures for Ireland, France, and Germany. In 1915, for pulmonary tuberculosis, the figures per 100,000 of the population run as follows: Scotland, 111; England, 116; Germany, 142; Ireland, 172; France, 179.

Such facts raise questions as to the view maintained by Metchnikoff and others that a process of gradual immunization is in progress in thickly populated countries through natural vaccination. With the figures before us, we are faced with the query: If some countries are thus reaping the benefit of prolonged natural vaccination, why are there such striking differences in the various groups forming the older civilizations? If natural immunization be the reason, why the difference in favour of Scot-

## TUBERCULOSIS FROM THE STANDPOINT OF PREVENTIVE MEDICINE

land over Ireland and France? If natural immunization be the reason, why should Scotland and England show the strikingly low mortality (111 and 116 respectively) as compared with France (179)?

**Incidence of tuberculosis.** (A) **In the population generally.**—There is accumulating evidence which goes to show that sooner or later everyone is tuberculized in less or greater degree. This would seem to be the explanation of such observations as those of Nägeli and Reinhart. The detailed analysis by the latter observer, showing that 32 per cent. of all bodies examined consecutively by him showed evidence of tuberculosis in presently aggressive form, illustrates strikingly the frequency of tuberculosis in its graver forms.

Such records raise the question: What proportion of this great incidence affords need for formal measures with regard to treatment and prevention? What proportion of persons in any given centre is likely to require medical surveillance with a view to treatment and prevention?

In an address delivered before the International Congress on Tuberculosis at Washington (1908) I expressed the view that most calculations as to the incidence of tuberculosis erred on the small side. Various computations led me to state that a minimal basis for assessing the incidence of tuberculosis requiring immediate attention in any district was obtainable by multiplying the recorded mortality from tuberculosis by 10; and further, that this multiple would be found to understate the actual fact, and I suggested that the multiple 20 might be adopted without exaggeration. Although the estimate has been criticized in different quarters, all subsequent experience has confirmed me in the opinion.

There is a growing consensus that the estimate is sound. Confirmatory evidence has been forthcoming from the United States. The results of the Framingham Community Health and Tuberculosis Demonstration (1916-18) and recent observations in Chicago affirm the fairness of the proposed multiple.

In addition to the numbers included in such an estimate, a still larger proportion of the community stand in need of more general preventive measures. It is obviously of importance for the successful elaboration of the scheme of anti-tuberculosis measures that we should have the working data as definite as possible.

(B) **Incidence in childhood.**—Statistics have accumulated rapidly which go to show that

tuberculosis is chiefly acquired in childhood. Evidence on the point, based on exact observation—clinical, pathological, and experimental—has been forthcoming from many sides.

1. *Clinical evidence.*—There is a consensus of opinion that congenital tuberculosis is rare. Even with tuberculous parents, the child is very seldom born with tuberculous disease. Whether the parents are tuberculous or healthy, the occurrence of tuberculosis in the child during the first six months of life is relatively infrequent. From that date onwards the frequency of tuberculosis increases steadily.

A number of years ago I submitted the statement, based on personal clinical examination of different groups of children of school age in Edinburgh, that at least 30 per cent. presented definite evidence of tuberculosis. With greater latitude in the interpretation of clinical signs, the percentage would have been larger. Later observations on the same lines, combined with the use of the tuberculin test, have satisfied me that the figure I have quoted was within the truth.

The evidence obtained from the system of the "march-past" of households, introduced by the Tuberculosis Dispensary, is striking. It is not uncommon to discover during one such examination signs of tuberculosis in three, four, or five children from the same house.

Since the introduction of the tuberculin cutaneous tests, numerous observations have been reported from different countries which all tend to the same conclusion, namely that tuberculosis exists, and can be determined individually, in the majority of school children. The figures of von Pirquet, Calmette, Ganghofer, Hamburger, Monti, and Franz go to show that in their respective cities most children among the working-class population have been tuberculized by the time they reach the age of 15.

Nor is the statement referable merely to the children of large cities. The observations of Franz included the examination of 400 young soldiers—apparently healthy recruits from the country, who had passed the ordinary physical examination for the service—and among these he found that 61 per cent. gave a positive reaction to the tuberculin test.

2. *Pathological evidence.*—The ultimate significance of the observations of Nägeli and Reinhart is apparent on turning to the post-mortem examination of children. Hamburger has reported the results in two separate groups.

## TUBERCULOSIS FROM THE STANDPOINT OF PREVENTIVE MEDICINE

The first series included 401 children dying from different diseases, of whom 63 per cent. of those between 7 and 10 years of age showed evidence of tuberculosis, and 70 per cent. of those between 11 and 14. The second series included 848 children, similarly dying from different causes. Of those between 7 and 10 years of age, 63 per cent. were found to be tuberculous, and 70 per cent. of those between 11 and 14. Deducting from the 848 those who had actually died from tuberculous disease, it was determined that of the remainder, between 11 and 14 years of age, no fewer than 53 per cent. had tuberculous lesions. Of special interest is the fact that in this series the frequency of tuberculosis steadily increased from 17 per cent. in those of 2 years of age to 53 per cent. in those between 11 and 14.

It is a point of much significance that during recent years, with increased refinement of methods, the number of children found to be tuberculous on post-mortem examination has steadily grown.

3. *Experimental evidence.*—Experimental research has shown that tuberculosis is of still more frequent occurrence than clinical and pathological observations have been able to determine. It has been proved that various conditions in which it had not been possible to discover characteristic appearances either at the bedside or at the post-mortem table were nevertheless of tuberculous nature. This has been determined in the case of young children, in whom no definite tuberculous lesion could be discovered in life or post mortem, with the exception of multiple infiltration of cervical and other glands. From these glands, in the absence of microscopical appearances of tubercle, it has been found possible to prepare an extract which, when injected into guinea-pigs, has produced tuberculosis.

From all this it will be seen that we are confronted by the fact that tuberculosis is vastly common in childhood. Most children take it at one time or another. The child's mucous membranes are specially receptive and absorbent. Inoculation occurs readily at any point. The extent of spread depends chiefly on the child's vitality or the resistance offered to the invading organisms by its living cells. This in turn is influenced largely by the character of the child's compulsory environment.

Other things being equal, the younger the child the more easy seems the dissemination of the virus throughout the lymphatic system. The frequency of infection increases in each

successive age-group. This is doubtless due to the continued, repeated opportunities for infection.

While, with advancing childhood, the frequency of infection increases, the morbid process progresses as a rule more slowly. Individual subjects differ much in this respect, but, other things being equal, the older the child the greater his resistance.

With increasing years the frequency of apparently arrested tuberculosis, e.g. in the form of calcified glands and scars, becomes noteworthy. Whether such arrest contributes in any degree to immunity is a difficult question. Some facts point in this direction. It is possible that some degree of immunization is established. If so, it is comparatively slight and fleeting. With continuous exposure to infection, under conditions of unsuitable environment, the immunity readily gives way, and the progress of the disease tends to be rapid.

(c) *Incidence in advanced age.* Tuberculosis—especially pulmonary tuberculosis—is to be found frequently in elderly persons. These are particularly the cases which escape notice. The disease is commonly of sluggish, undemonstrative type. Apart from chronic cough and expectoration, with some dyspnoea, there is little calling for attention. It occurs in the grandparents and other elderly relatives of a household, who are commonly spoken of as bronchitic or asthmatic. Yet, if examined, a large proportion will be found to present physical signs of disease and a not inconsiderable proportion expectorate tubercle bacilli. These old people must unhappily be regarded as carriers of the disease to children in the household and to others with whom they may be in frequent close contact.

*Variety of clinical manifestation.*—No other disease is so kaleidoscopic in its expression. There is hardly a structure or organ of the body which it may not invade. In some cases the infiltrative process may remain comparatively slight—it may be latent. Apart from these, the progress of the disease varies endlessly from cases which run a rapidly fatal course, measured by weeks, to cases which may continue throughout a long lifetime. There are cases of "open" tuberculosis in which bacillus-containing discharge is present, and cases of "closed" tuberculosis without such discharge. Of special significance is the distinction to be drawn between cases where the process, however extensive, remains for the

## TUBERCULOSIS FROM THE STANDPOINT OF PREVENTIVE MEDICINE

most part local, and cases where the local process, whether slight or pronounced, is associated with systemic disturbance.

**Clinical classification.**—Attention has hitherto been too much directed towards the local manifestations of tuberculosis, whether occurring in the lung or other organ. Most clinical classifications of tuberculosis suffer on that account.

To be of scientific and practical value, clinical classification must include an estimate not only of the amount of anatomical involvement but also of the degree of systemic disturbance.

The so-called Turban-Gerhardt classification of pulmonary tuberculosis, which was adopted by the International Conference on Tuberculosis in Vienna, suffers from the disadvantage that the classification is based almost entirely on the amount of anatomical involvement within the lung. Little cognizance is taken of systemic disturbance. The three stages of pulmonary tuberculosis recognized under that scheme are as follows :—

*Stage I.*—Disease of slight severity, limited to small areas of one lobe, which, for example, when affecting the apices bilaterally, does not extend beyond the spine of the scapula and the clavicle, or, unilaterally, does not extend below the second rib anteriorly.

*Stage II.*—Disease of slight severity, more extensive than Stage I, affecting at most an entire lobe, or of greater severity, extending at most over half a lobe.

*Stage III.*—Disease of greater extent than just defined, and all cases with considerable cavities.

Such a classification, excellent as it may look on paper, is misleading and insufficient. In actual fact, a case in Stage III may really be less serious than a case in Stage I, and so on. The classification does not differentiate between the various types of illness which anatomically may have more or less the same basis.

The following classification gives approximately just expression both to the amount of *anatomical involvement* and to the *systemic disturbance*. It groups cases more satisfactorily from the clinical standpoint, and affords valuable indications as to prognosis and treatment.

The symbol **L** represents the local or, in the case of pulmonary tuberculosis, the lung lesion, and the symbol **S** the systemic disturbance. For convenience, the three anatomical stages, already referred to, are accepted and described

as **L<sub>1</sub>**, **L<sub>2</sub>**, **L<sub>3</sub>**. By the simple device of combining variously capital and small letters, the diagnosis at a given time can be expressed with reasonable accuracy. Thus, in cases with a limited involvement of the lung, the various possibilities may be stated as **L<sub>1</sub>**—a slight local process without systemic disturbance; or **L<sub>1</sub>s**—a slight local process with relatively slight systemic disturbance; or **L<sub>1</sub>S**—a slight local process with equivalent systemic disturbance; or **l<sub>1</sub>S**—a slight local process with excessive systemic disturbance. It matters not what amount of *local* lung involvement exists, whether **L<sub>1</sub>**, or **L<sub>2</sub>**, or **L<sub>3</sub>**, the same principle of classification is available.

Practical experience has shown the value of such a scheme. It has not merely a value in relation to diagnosis at a given moment, but affords a most serviceable means of *recording the changes which occur from time to time* in such cases, whether in the direction of improvement or the reverse.

The following table shows the scheme of classification and the symbols in use :—

<b>L<sub>1</sub></b>	<b>L<sub>1</sub>s</b>	<b>L<sub>1</sub>S</b>	<b>l<sub>1</sub>S</b>
<b>L<sub>2</sub></b>	<b>L<sub>2</sub>s</b>	<b>L<sub>2</sub>S</b>	<b>l<sub>2</sub>S</b>
<b>L<sub>3</sub></b>	<b>L<sub>3</sub>s</b>	<b>L<sub>3</sub>S</b>	<b>l<sub>3</sub>S</b>

The presence of complications is indicated by the given symbol followed by a reference to the lesion, e.g. **l<sub>3</sub>S** + *ent. tub.* indicates a case of extensive lung involvement (vomica formation) and excessive systemic intoxication, along with intestinal tuberculosis.

**Tuberculosis in households.**—That tuberculosis is a disease of households is one of the oldest observations in medicine, the full significance of which has but recently been apprehended. In former times the observation led to the erroneous view that tuberculosis is hereditary.

Two questions present themselves for consideration in every instance: (1) Is there an existing source of infection within the household? (2) Are the environmental requirements for a healthy life and effective resistance against tuberculosis available in the home?

(1) An existing source of infection within

## TUBERCULOSIS FROM THE STANDPOINT OF PREVENTIVE MEDICINE

the household may be present in a readily recognized consumptive, or even more frequently in a member of the household hardly suspected of being tuberculous, e.g. the elderly relative with so-called "bronchitis." Or the source of infection may be the house itself, its walls, hangings, furniture, or floors.

It was with a view to reach such possibly hidden source of infection and to gauge accurately the degree of dissemination of infection throughout the household that many years ago I was led to institute, in connexion with the Tuberculosis Dispensary, the domiciliary visit and the systematic "march-past" of the household. It was not uncommon to discover the primary source of infection in a parent or other relative who was not regarded as ill, and to discover three, four, or even more children already infected in the household.

(2) The question of environment involves considerations as to the site and structure of the house, the amount of accommodation in proportion to the number of inmates, the freedom with which air and sunlight reach the apartments, the sufficiency and suitability of food supplies, clothing, and generally the understanding, or otherwise, of household management.

Here again the key to the situation was afforded by the domiciliary visit of doctor and nurse—going back, so to speak, with the tuberculous patient to his home and studying his environment, in the widest sense, day and night.

One result of intensive anti-tuberculosis activity has been to accumulate a large collection of facts which incriminate the home as the nursery of tuberculosis. The Edinburgh records have shown that in 60 to 70 per cent. of the cases under the care of the Tuberculosis Dispensary there existed all the possibility of infection—that is to say, persons living in the given household had been for prolonged periods in closest proximity to one case, at least, of pronounced tuberculosis, and in more than 65 per cent. of the cases the tuberculous patient occupied the same bed with one or several persons; and that in more than 75 per cent. of the cases the tuberculous patient occupied, if not the same bed, at least the same room, and that 70 per cent. of the cases occurred in dwellings consisting of not more than two rooms; in a large number of instances the house was otherwise insanitary.

**Susceptibility.**—There is a growing con-

sensus of opinion, based on clinical and pathological observations (both in man and in animals), that tuberculosis is *not* hereditary in the strict sense of the word. The offspring of tuberculous parents is very seldom born tuberculous. It comes into the world for the most part free from tuberculosis. The fact in relation to cows has been long recognized and is the basis of the preventive treatment of tuberculosis among cattle, introduced in Denmark, known as the Bang system. If a calf be removed immediately at birth from contact with its tuberculous parent and transferred to a fresh, non-infected cow-house, and due care be given to its rearing, the animal remains free from tuberculosis. Contrariwise, calves left after birth in contact with diseased mothers and reared by them commonly develop tuberculosis.

Tuberculosis is acquired by the offspring in proportion as it is kept in close propinquity to the potential source of infection in the parent, under conditions of environment which make it difficult for the tender, succulent, and absorbent tissues to withstand inoculation. In proportion as those factors are excluded, the offspring remains free from infection.

While this is true, it may well be that tuberculous parents pass to their progeny a lowered power of resistance to the disease. An hereditary transmission of less resistant tissues is not excluded. Relative susceptibility or insusceptibility to certain other diseases has been recognized in individuals, families, and races. In bacteriological research the insusceptibility of certain animals to certain contagions and poisons is well known. In the department of therapeutics, certain methods of treatment are based on the recognition of the relative insusceptibility of certain animals.

We are not entitled to predicate more for the influence of heredity on tuberculosis than the production in the tissues of the offspring of a susceptibility to, or a diminished power of resisting, the tubercle bacillus.

**Prevention and control.**—The problem of the prevention and control of tuberculosis is far-reaching and difficult. We have to deal with an infective process of slow, insidious nature, for the most part, which is highly tractable in the earlier stages, readily arrested under suitable conditions of environment, but correspondingly persistent in the absence of these. Further, we have to deal with a disease with immensely wide ramifications throughout the community, to which everyone is liable



## TUBERCULOSIS FROM THE STANDPOINT OF PREVENTIVE MEDICINE

and with which, sooner or later, the majority becomes affected in some degree.

The measures to be followed divide themselves naturally into—

1. Prevention.

2. Control and treatment of existing cases.

1. **Prevention.**—This includes (1) measures directed towards increase of resistance on the part of the individual and the community—the creation, broadly speaking, of suitable environment, and (2) the removal of sources of infection.

(1) *Personal measures.*—From infancy onwards the citizen must be bred in an environment which makes for resistance. The great practical difficulty lies in the ignorance and helplessness of the average individual and household in respect of air, cleanliness, and food.

Effective resistance will follow in proportion as the vitalizing properties of fresh air and cleanliness are understood and demanded in the ordinary household, and the child's compulsory environment is maintained in accordance with physiological principles which make for health. These principles are simple and broad, and their application is easy and costs little.

There is urgent need for education in the laws of health. From childhood onwards man and women must be trained to "know themselves." Beginning with the simplest possible facts, they should be made to understand the machinery of the human body and the fundamental principles of health. In this direction much might be achieved by doctors, nurses, health visitors, and social workers.

Were the cardinal principles of the sanatorium realized within the home and workshop and in communal life generally, the influence of the tubercle bacillus might be largely discounted. Every school should be an object lesson in health methods, and first and foremost of such object lessons should be the constantly open window. Every school should be an open-air school. School teachers must realize for themselves, and so be able sympathetically to impress on their scholars, the paramount significance of oxygen in healthy life.

There must be practical training in domestic economy—object lessons in the selection and preparation of food and clothing, cleansing of the dwelling, washing, lighting, in the ready detection of departures from health and in the general principles of nursing.

The main facts regarding the occurrence and propagation of tuberculosis should be common knowledge. Everyone should understand that tuberculosis is not got by exposure to cold, that, contrariwise, the more freely the individual enjoys fresh air and sunlight, the more certainly will he avoid tuberculosis.

Dusting and sweeping of rooms and offices ought to be carried out by moist methods, and, when possible, immediately after, rather than before, occupation.

In choosing a house, its potential of air and sunlight should be the first consideration. Dryness of the residence is essential.

Every effort must be made to lead the physiological life. Etiological facts all go to show that the more the individual is in the open air, the less is he likely to contract tuberculosis. Contrariwise, the more he is indoors, the greater is the risk. This risk must be minimized as far as possible for those whose business is chiefly sedentary by the direct admission of fresh air during working hours and by an extra allowance of fresh air at other times, especially by night. The sedentary habit must be counterbalanced by activity in the open in off times. Respiratory movements and postural exercises—in a word, lung gymnastics—should be practised regularly, either in the open-air or, when this is impossible, in a room with the window freely open.

Life from day to day should be regular and simple. The toilet of the skin (baths, rubbing, etc.) should be carefully attended to. Clothing, while sufficiently warm, should be light, and all constriction excluded. Chest protectors and extra mufflers should be taboo. The use of stimulants and tobacco, if indulged in, should be carefully regulated, and cigarette smoking should be excluded.

After acute illnesses, particularly measles, whooping-cough, and influenza, special care must be taken to ensure satisfactory convalescence.

*Safeguards against infection.*—In view of the risk of infection through milk, where there is any doubt as to the purity of milk supply all milk taken by children should be scalded before use.

When one of the household is already affected, it is necessary to adopt certain precautions to safeguard others. The essential precautionary measures are simple and easily carried out. If this be done, the presence of the tuberculous patient need not be regarded as a serious menace. The breath of the con-

## TUBERCULOSIS FROM THE STANDPOINT OF PREVENTIVE MEDICINE

sumptive patient does not convey infection. The risk of carriage of infection in coughing—which is probably less than some authorities suppose—may be readily avoided by screening or the like. As the chief danger lies in the expectoration, this should be carefully discharged into a jar or flask containing a tablespoonful of disinfectant, or into a destructible handkerchief.

The tuberculous patient should not share the same bed, or, indeed, the same sleeping room with another. This applies with special force in the case of the tuberculous mother. An affected mother should not suckle her child. Kissing and fondling should be avoided.

The subject of tuberculosis should not marry when there can be any doubt as to the absolute arrest of the disease. The physician should not give his sanction to marriage unless, under definite observation, the tuberculous process has been perfectly quiescent for a year or two. It is frequently well to extend the probationary period.

The tuberculous patient must be jealously careful not to swallow expectoration. Swallowing means the risk of carrying bacilli to other organs.

Unhealthy conditions of tonsils and enlargement of lymphatic glands should be watched with much care.

(2) *Public measures.*—Every community must insist on the maintenance of sanitation on a high level. Local authorities have large powers in this direction, and they must be urged to use them.

Wisely-devised measures for the increase of sunlight and air throughout cities and towns do much in the prevention of tuberculosis. This includes the removal of insanitary quarters, the provision of open spaces, gardens, and parks, and the widening of streets.

Housing reform is imperatively needed. It is obvious that every man, woman, and child must have a satisfactory home, sufficient in every direction to meet the physiological needs of healthy existence and development.

The inspection of lodging-houses, workshops, factories, places of assembly and public conveyances should be more stringent. Cleanliness of streets must be insisted on, and street cleaning should be rendered harmless by watering the streets previously to brushing them. Spitting on the streets and in assembly places and public conveyances should be forbidden as a nuisance.

Sufficient powers must be given and used for

the supervision of the milk supply of the area of population.

Under the Ministry of Health in England and Wales the following bodies are concerned, in varied degree, in dealing with tuberculosis, viz.: (a) County Councils, (b) Sanitary Authorities and Joint Hospital Boards, (c) Insurance Committees, (d) Local Education Authorities, (e) Poor Law Authorities, (f) the Metropolitan Asylums Board. Under the Board of Health in Scotland the authorities concerned include (a) for Boroughs the Town Council, (b) for counties divided into districts the District Committee, (c) for counties not divided into districts the County Council, (d) Insurance Committees, (e) Local Education Authorities, (f) the Parish Council.

In respect of persons discharged from the Services because of tuberculosis attributable to, or aggravated by, such service, the Pensions Ministry has important responsibilities. These are fulfilled by relations established between the Pensions Ministry and the Health Ministry (Insurance Commissioners), and locally by Insurance Committees acting in relationship with Local War Pensions Committees.

In addition to these authorities, voluntary bodies have, in varying degree, undertaken work and maintained institutions in relation to tuberculosis. These include the National Association for the Prevention of Tuberculosis, the Central Fund for the Promotion of Dispensaries in London, the Committees of the various Tuberculosis Hospitals throughout the country, and the Royal Victoria Hospital Tuberculosis Trust (Scotland).

The legal measures under which the various authorities act include especially the Public Health Acts of the several divisions of the country and successive Tuberculosis Regulations, the Infectious Disease (Notification) Act (1889), the Isolation Hospitals Act (1893), and the National Insurance Act (1911) and successive Tuberculosis Regulations.

### 2. Control and treatment of existing cases.

—These imply a knowledge of the incidence and distribution of tuberculosis throughout the country, and the establishment of suitable and sufficient machinery for dealing with these.

**Anti-tuberculosis measures.**—It is obvious that in framing an effective anti-tuberculosis scheme, it is essential to combine measures of prevention with measures for the care and treatment of individual cases.

If the ultimate outlook of the Department of Health is towards the prevention of tuber-

## TUBERCULOSIS FROM THE STANDPOINT OF PREVENTIVE MEDICINE

culosis, it is no less clear that for a long time to come it will have to deal directly with the treatment of a great mass of tuberculous material. It is foolish to speak—as is frequently done in certain quarters—as if the one solution of the tuberculosis problem were to be found in better housing of the people.

The provision of better houses admittedly plays an important part in the correction of faulty environment, and this has been considered above.

The tracking of tuberculosis to the home was one of the earliest triumphs of the Tuberculosis Dispensary. The following of the tuberculous patient back to his "nest" and studying the conditions of the home and the household largely contributed to the exposure of the clamant need for an improvement of immediate environment. It was the domiciliary visitation of cases which bore most piquant testimony to the crying shame of much that passed for human habitation.

The discovery in the sanatorium of the extraordinary influence exerted on tuberculosis by the institution of a physiological environment emphasized the contrast between healthy environment and that present in a large proportion of the homes of the community. The sanatorium afforded the proof that, if only the proper environment could be reproduced in the homes of the people—both in town and in country—the incidence of tuberculosis would be enormously reduced.

But it is no less evident that the millennium cannot be realized for many a day. Meanwhile, tuberculosis reproduces itself in countless instances.

The source of infection remaining, the recurrent cases of tuberculosis in every community will have to be dealt with.

An effective *anti-tuberculosis organization* must have an outlook on all sides of the question.

1. It must have in view the etiological factors which lead to the production of the disease, including both environment and sources of infection.

2. It must recognize the most effective measures for combating these and preventing disease.

3. It must have available means for the estimation of the incidence of the disease and for its detection at the earliest possible moment.

4. It must render available to the community generally the best methods of treatment

for the varying forms and stages of the disease, either in institutions or at the patients' homes.

5. It must endeavour by a suitable propaganda to educate the community regarding the causation, prevention, detection, and treatment of the disease.

6. It must insist on a more special training of the medical profession (doctors, nurses, etc.) in the prevention, diagnosis, and treatment of tuberculosis.

7. It must seek to turn the experience gained in the working of the scheme to more general use by collating results and by encouraging research with regard to the many-sided aspects of the disease.

It is clear that any anti-tuberculosis scheme will be effective only in proportion as it is based on the facts and needs of the case. In its essentials, an effective tuberculosis scheme should be applicable throughout an entire country. It might, indeed, be uniform throughout the world.

**Tuberculosis Schemes.**—It was considerations of the sort just enumerated which led the British Departmental Committee on Tuberculosis (1912-13) to propose the erection of machinery for combating tuberculosis. Such machinery is now largely stereotyped in the so-called Tuberculosis Schemes in the United Kingdom.

The principle embodied in the Model Scheme reproduces what had been evolved in the practical solution of the problem from 1887 onwards, and had come to be known as the Edinburgh Scheme.

The essentials of the scheme are —

1. Notification.
2. The Tuberculosis Dispensary with its varying activities.
3. The Sanatorium for the treatment of suitable cases with a view to arrest of the disease.
4. The Hospital with various possibilities for the treatment of acute conditions, segregation of advanced and dying cases, education of more chronic cases, etc.
5. The Working Colony for more continuous treatment on simpler lines of hopeful cases requiring more prolonged care with concurrent training.
6. Care Committees concerned with the social side of the problem, and the oversight of the patient from the economic point of view.

## TUBERCULOSIS FROM THE STANDPOINT OF PREVENTIVE MEDICINE

It is desirable to review each of these factors in somewhat greater detail.

1. **Notification.**—In a disease like tuberculosis, of infective character, varying manifestation, and widespread distribution throughout the community, it is obviously desirable that the Health Authorities should be in possession of as complete information as possible regarding its incidence.

It is all the more remarkable that, notwithstanding the advocacy of those who knew most about the subject and were therefore best entitled to make the demand, it took a long time before notification of tuberculosis became general.

The first successful move occurred in New York where, in 1893, notification "was *required* of cases of consumption in all public institutions, and was *requested* voluntarily from doctors of cases occurring in their private practice." The measure worked so satisfactorily and smoothly that in 1897 the Health Board of New York declared pulmonary tuberculosis to be "an infectious and communicable disease" and *required* "notification of all cases of consumption occurring in the city."

In the United Kingdom, following much discussion, voluntary notification was gradually introduced in a number of cities. The chief difficulty in the way of compulsory notification was the attitude of reserve adopted by the Local Government Boards. Finally, however, in 1912 notification of pulmonary tuberculosis was made compulsory, and in 1914 all forms of tuberculosis became notifiable. The doctor in attendance on the case was made responsible for the notification, and a small fee was paid to him in respect of each case notified.

Much has been made of the difficulties of notification of a disease so varied in its manifestations as tuberculosis. It should be kept in view, however, that apart from detection of tubercle bacilli, there are abundant grounds for the diagnosis of a large proportion of cases. This is particularly true in more advanced stages, where it is especially desirable to have notifications as complete as possible.

It is clear that from one cause or another many cases remain unnotified. But the existence of the demand has made examination more careful and thorough. Practitioners have become more accurate in the diagnosis and record of their cases. Mistakes continue to be made, commonly in the direction of omission.

With growing knowledge of the disease and refinement of methods of diagnosis, notifications will tend to become more exact and exhaustive.

*Relation to the Public Health Department.*—Notifications are made officially to the Medical Officer of Health. Where a satisfactory Tuberculosis Scheme is in operation, they are passed to the Tuberculosis Officer, who is the responsible representative of tuberculosis in the Department.

A good deal of unnecessary trouble and irritation has arisen over the relation of the Medical Officer of Health to the subject. So long as there is one official head to the Health Department of a Local Authority, and he is designated the Medical Officer of Health, it is convenient that reports regarding tuberculosis, as regarding other diseases, should be sent to him. In this sense he is the Administrative Officer. Within the Public Health Department, however, if it is properly organized, there should be a Tuberculosis Department manned by Tuberculosis Officers of varying grades. The chief of these executive officers should receive the notifications and deal with them practically as the circumstances demand. In numerous instances he may not require to be in clinical relationship with the patient notified. In those cases where he should be in close touch there is available for the purpose all the machinery which is represented by the Tuberculosis Dispensary.

Following notification, the Tuberculosis Department may think it desirable to make a domiciliary visit for the purpose of assuring that immediate environment is satisfactory. In such a case the executive officer will get into communication with the notifying doctor. In some instances it will be found that the notifying doctor will be very glad if further responsibility for the care and treatment of the case be transferred from himself to the Tuberculosis Department. The further steps to be taken will be considered under the head of The Tuberculosis Dispensary.

2. **The Tuberculosis Dispensary.**—The purpose in establishing the tuberculosis dispensary was to create a *centre* which should concern itself with every aspect of the tuberculosis problem.

The dispensary was projected as a central institution or bureau in respect of all the tuberculous material in the district. This central institution would draw to itself persons affected

## TUBERCULOSIS FROM THE STANDPOINT OF PREVENTIVE MEDICINE

by tuberculosis or who might be suspected by themselves or their doctors to be so suffering. It would constitute an "information bureau" in respect of all inquiries concerning tuberculosis, and a "supervision bureau" in respect of patients who did not require hospital treatment, but where home supervision and general direction were desirable. It would serve as a "clearing house" in respect of the different types of tuberculosis which presented themselves and which might require institutional treatment. It would form the *nodus* or connecting-point of diverse measures which had for their common purpose the prevention and treatment of tuberculosis.

The first tuberculosis dispensary—the Victoria Dispensary—was established in Edinburgh in 1887. The programme of operations was as follows :

1. The reception and examination of patients at the dispensary, the keeping a record of every case, with an account of the patient's illness, history, surroundings, and present condition; the record being added to on each subsequent visit.

2. The bacteriological examination of expectoration and other discharges.

3. The instruction of patients how to treat themselves, and how to prevent or minimize the risk of infection to others.

4. The dispensing of necessary medicines, sputum bottles, disinfectants, and, where the patient's condition seemed to warrant it, food-stuffs and the like.

5. The visitation of patients at their own homes by (1) a qualified medical man, and (2) a specially trained nurse, for the double purpose of treatment and of investigation into the state of the dwelling and general conditions of life and the presence of infection in others.

6. The selection of more likely patients for hospital treatment, either of early cases for sanatoria or of late cases for incurable homes, and the supervision, where necessary, of patients after discharge from hospital.

7. The guidance, generally, of tuberculous patients and their friends, and the answering of inquiries from all interested persons on every question concerning tuberculosis.

The *methods* of the tuberculosis dispensary are entirely different from those of the ordinary out-patient department. This cannot be too much emphasized. The dispensary views tuberculosis as an infective and endemic disease, ramifying throughout the social organization

in quite unique fashion. Tuberculosis reflects and, in turn, influences social conditions in extraordinarily varying ways.

The dispensary recognizes the unity of tuberculosis in all its stages and in all its remarkably varied manifestations. Its aim is that not a single case of tuberculosis shall occur unobserved, or remain uncared for, in the community.

Once it gets in touch with an individual patient, it never loses hold of him. Either it treats him directly, or it sends him for treatment to the appropriate institution. In the latter case it undertakes his after-care when institutional treatment is over.

Beyond all this, it seeks to *discover tuberculosis in its recognized haunts*. One of its most valuable functions is to trace the patient to his home—to get at what I have termed the "tuberculous nests," to investigate faulty environment, and to search out tuberculous disease among the other members of the household, and in turn to undertake the care of these. It takes the home and household in hand, so far as it may be infected by tuberculosis.

Every tuberculous individual coming within the cognisance of the dispensary is dealt with (a) in his own interest and (b) in that of the community.

Personally, the sick man receives the form of treatment appropriate to his case, either in his own house or at the dispensary, or in one of the institutions linked with the dispensary—the treatment being directed and maintained until all risk is past.

From the point of view of the community, the patient leads the way to the home and the household. While obviously looking after the sick man, the dispensary takes occasion to study his surroundings and to search for other cases. This is attained by visits of (1) the dispensary nurse and (2) the dispensary doctor, which follow in every instance. It is of first moment that these visits should be undertaken by skilled members of the dispensary staff. Thereby the work is done uniformly and efficiently, and risk of overlapping is avoided.

Where the home conditions are such as to call for structural alteration of the dwelling, or, it may be, its condemnation, the matter is referred by the dispensary doctor to the medical officer of health, just as would be done in similar circumstances by a private practitioner.

## TUBERCULOSIS FROM THE STANDPOINT OF PREVENTIVE MEDICINE

In this way the tuberculosis dispensary becomes the centre of anti-tuberculosis activity within the given area, and the connecting link of an organized system of anti-tuberculosis agencies which include the sanatorium, the tuberculosis hospital, the farm colony, the tuberculosis school, etc.

In relation to notification, the dispensary plays a twofold part. In the first place, it constitutes an important notification agency. The dispensary attracts tuberculous patients at all stages, who would in many cases either not consult a doctor, or might present themselves for advice under conditions where the detection of disease is less likely. By its system of examination of homes and households it discovers tuberculosis at the earliest point. Thus, of cases notified during 1912 (before tuberculosis was notifiable) in the borough of Kensington no fewer than 61·8 per cent. were notified by the tuberculosis dispensary, and similarly in the borough of Paddington 40·3 per cent. were notified by the dispensary. In Edinburgh more than 50 per cent. of the cases notified under compulsory notification of pulmonary tuberculosis have been notified by the dispensary.

After notification the dispensary once more steps in and decides in practical fashion what is to become of the notified individual. The tuberculosis dispensary takes and keeps hold of the tuberculous patient in his own interest and that of the community.

While the dispensary must be linked closely with every agency concerned with the oversight and care of tuberculous cases, it must be linked likewise with the general body of practitioners, to whom is committed the treatment of tuberculous patients at their own homes. The resources of the dispensary should be placed at the service of the doctors, for the purpose either of consultation or of treatment. The expert staff of the dispensary should be available to give advice on any question in relation to tuberculosis. Inquiries should be welcomed on all subjects which relate to the disease.

*Reception of patients.*—The benefits of the dispensary must not be too strictly limited to *admittedly* tuberculous subjects. Special care must be practised as regards suspect cases. The doubtful case of to-day is frequently the definite tuberculous patient of to-morrow. With growing experience and refinement of methods, the thorough clinician finds that the

number of patients merely “suspect” diminishes while the number diagnosed as “tuberculous” increases.

All patients must be aroused to a personal interest in the care of their health and the treatment of their disease. By quiet talks—*causeries*—to groups of patients, and the regular training of them in physiological methods, much is to be gained. The patient's interest is awakened, and he becomes the main agent in his cure.

*Examination of “contacts.”*—This is one of the most important of the dispensary functions. The tuberculous seedling is discovered at a stage when it can be readily dealt with. To be effective, the work should be in the hands of *the ablest and most experienced* of the staff. The work must be thorough and discriminating. The earliest stigmata of tuberculosis must be appreciated. When a “contact” is recognized as tuberculous, he must be placed under regular supervision and his condition watched as carefully as if he were more gravely affected.

*Repetition of examination.*—The examination of contacts must not be limited to one occasion only. Contacts who at the first examination are passed as non-tuberculous must be reviewed from time to time. Cases about which there is doubt should be re-examined more frequently, say every three months. So long as the primary source of infection remains in the home, the possibility of successful infection of others continues; indeed, the chance of successful inoculation increases. It is a good rule, in case of death of the original case or removal to sanatorium or hospital, to make a specially careful revisit of the other members of the household.

*Domiciliary visitation.*—The thorough investigation of the home and the household is of first importance. Without a systematized plan of domiciliary visitation by doctor and nurse, a large part of the value of the dispensary is lost. If the dispensary nurse be worthy of her position and training—she is something more than a mere health visitor—she will readily win the confidence of the patients by her interest in their welfare. She instructs the patients and their friends (wives, mothers, etc.) as to treatment and prevention. In co-operation with the visiting doctor, she should report regarding the patient's environment. The lines of inquiry are suggested in the accompanying schedule of inquiry.

# TUBERCULOSIS FROM THE STANDPOINT OF PREVENTIVE MEDICINE

## SCHEDULE OF INQUIRY REGARDING DISPENSARY PATIENTS

No. in Ledger ..... Date of Report.....

Name ..... Married or single ? .....

Address .....

Occupation ..... Has patient changed occupation ? .....

Able to work full time ? ..... Or part time ? .....

If unable, confined to bed ? .....

How long ill ? .....

Situation of house (area, ground floor, first, etc.) ? .....

Number and ages of inmates ? .....

Number and description of rooms ? .....

General aspect of house (clean, damp, dusty, smelly) ? .....

Number of windows ? ..... Can they open ? .....

Are they kept open (a) by day ? .....

(b) by night ? .....

Have they always been kept open ? .....

Does patient sleep alone (a) in bed ? .....

(b) in room ? .....

How is washing of clothes done ? .....

How long in present house ? .....

If has moved within two years, previous addresses ? .....

Have there been illnesses or deaths in house ? .....

(a) In own time ? .....

(b) In previous occupancy ? .....

Exposed to infection (a) at home ? .....

(b) at work ? .....

(c) among friends ? .....

Present health of other members of household ? .....

What precaution taken to disinfect ? .....

T. B. in sputum ? .....

T. B. in dust of room ? .....

General dietary ? ..... Tectotal ? .....

General condition (well-to-do, badly off) ? .....

Proximate income of household ? .....

Assisted by societies, church, friends, rates ? .....

(Signed) ..... Reporter.  
 ..... Medical Officer.

*Records.*—The record regarding each patient should be complete and continuous. His *dossier* should include an account of his history, condition, and progress, his home conditions, his relationship to work. The physical condition of patients should be reported according to a definite and uniform scheme, supplemented, where desirable, by an X-ray photograph.

Where a patient passes to an institution, a specially careful note should be made of his actual state, and, on discharge from the institution, a summary of his treatment and progress during residence should be added. Thereafter a summary of his progress should be made from time to time.

*Statistical department.*—Each dispensary

## TUBERCULOSIS FROM THE STANDPOINT OF PREVENTIVE MEDICINE

should have its statistical department. This tends to ensure uniformity and continuity of record. If properly worked, it affords a reliable and up-to-date Directory (Address-book) of Tuberculosis for the areas served by its operations. This accumulated information in any direction can be readily tapped.

To be effective, the scheme of records should be as simple and direct as circumstances will allow. By means of well-devised card-indexes it is easy to collate in a short time the facts bearing on any line of inquiry.

*Publication and propaganda department.*—It is the duty of the dispensary to keep alive the public interest in the prevention and treatment of tuberculosis. This can be readily effected by means of attractive cards and leaflets on such subjects as, *How to avoid infection, How to keep fit, How tuberculosis is spread, How to treat tuberculosis in a household*, and so on. From time to time popular lectures and addresses, illustrated by lantern slides and cinema, may be given on similar subjects.

*Examination of sputum.*—Patients coming to the dispensary must be enjoined to bring—preferably in a jar given them for the purpose—a clean specimen of expectoration or other discharge for examination. The results of such examination, whether positive or negative, will be added to the *dossier*.

If the examination is negative, care must be taken to ensure a sufficiently frequent repetition of the test. The routine of such examination, if properly systematized, is not burdensome. It is easy to train a lay assistant or laboratory boy to do the mechanical part of the process.

**3. The Sanatorium.**—The sanatorium has for its special purpose the effective arrest of the disease in curable cases and the education of the patient and the public in its treatment and prevention.

Nature cures tuberculosis every day. The aim of sanatorium treatment is to assist Nature in her effort to resist invasion. The purpose is to increase vital resistance and so establish more or less immunity.

The essentials of the sanatorium are free exposure of the patients to open air, proper dietary, perfect rest when necessary, at appropriate stages carefully regulated alternation of rest and movement, suitable clothing, and attention to the skin—a *physiological* regimen in the largest sense. In addition, the sanatorium affords opportunity for the continued application—under close medical observation—

of specialized lines of treatment, e.g. tuberculin, operative measures, etc., which might be otherwise impossible.

*Selection of cases.*—Success in sanatorium treatment largely depends on the care and skill with which cases are selected. Broadly speaking, the earlier treatment is undertaken, the more likely is it to be effective. A large part of the value of sanatoria has been lost by the lack of exact care in the selection of the suitable patient.

Sanatoria have been blocked by a crowd of wholly unsuitable patients. They have been allowed to become dumping-grounds for every sort, and the undesirable (incurable) has been especially in evidence. The result has been hopeless confusion, the piling up of long waiting lists, and the exclusion, at the proper moment, of suitable cases, with the inevitable result that in many instances the suitable have perforce lapsed into the unsuitable. In this way an interminably vicious circle has been established.

*Site of the sanatorium.*—A great mistake has been commonly made in the choice of site. Sanatoria have too often been placed in positions, desirable enough in themselves, which are hopelessly inaccessible and otherwise inconvenient. They ought to be established in *immediate relationship with the large centres*. The point cannot be emphasized too much. It is of primary importance, for the larger solution of the problem, that patients and the population generally should get rid of the prevalent, erroneous belief that a cure can only be effected and health maintained under conditions which their ordinary residence and position will not allow them to enjoy. It has been demonstrated over and over again that, just as tuberculosis occurs in all climates, so it can be treated in all climates with approximately equal success, provided that the physiological indications for treatment are fulfilled. That being so, it is obviously an enormous advantage that the sanatorium should be in reasonable proximity to the centre of population which is chiefly interested.

*Results of sanatorium treatment.*—The value of sanatorium treatment to individual tuberculous persons and as an object lesson in the prevention and treatment of tuberculosis is acknowledged by the great majority of those whose experience entitles them to speak. Given a well-managed sanatorium and suitably selected patients and a sufficient length of residence, the value of the sanatorium cannot



## TUBERCULOSIS FROM THE STANDPOINT OF PREVENTIVE MEDICINE

be exaggerated. Everything depends on the conditions that have been premised. There are sanatoria and sanatoria. Some are unworthy of the name. Sometimes innocently, sometimes perversely, most unsuitable cases have been thrust even into suitable sanatoria, and there has been little attempt to measure the length of treatment to the needs of the case. The limit of residence has been fixed by rule-of-thumb methods for economic or other reasons. The result has been incomplete, and an incomplete result in tuberculosis commonly means failure.

The lesson has yet to be learned by most local authorities that *tuberculosis takes a long time to come and no less certainly takes a long time to go*. The random, thoughtless judgment that is meantime current that sanatorium treatment has proved a failure is *not* justified by facts.

In the Tuberculosis Scheme the sanatorium was established to effect a special purpose in the campaign—namely, the treatment of *early* cases. It was not projected for the patching up of damaged lives. Its use for the latter class has been the result of muddling on the part of those who, without training and patience to understand the issues, have adopted the easy creed that “One man’s guess is as good as another’s.” It is hardly fair to complain of the failure of machinery to do work for which it was not intended, nor is it sound to propose to scrap machinery which, far from having failed, has not for the most part been given a chance.

**4. The Hospital.**—There are cases of tuberculosis beyond the reach of recovery. Some of these are of highly chronic character and relatively unimportant. On the other hand, there is a large group of advanced and progressive disease. This constitutes the greatest source of danger to the community. The risk of infection when such patients are resident in the contracted dwellings of the poor is very great. It is especially for such that hospitalization is clamantly called.

What is wanted is that the patient should be nursed and cared for in a satisfactory manner without prejudice to the members of his household. Fortunately the number of such cases tends to diminish steadily in proportion as the operations of the other factors in the Tuberculosis Scheme—especially the Tuberculosis Dispensary—are satisfactorily developed.

The hospital may either be a separate in-

stitution specially devoted to the purpose, or may conveniently be part of a general infectious hospital. Whichever form it take, it is most desirable that the hospital should be regarded both by doctor and by patient as a hospital for the treatment of the disease, and not a mere asylum or segregation centre.

Patients must be encouraged to come into hospital with the hope of more skilled treatment than is possible at home. They should be advised by every possible means to remain in the hospital until the end, or until, as sometimes happens, they are so obviously better that they may be suitably transferred to another institution or to their own home. If the hospital is to fulfil its purpose, from the preventive point of view, patients in an advanced stage of the disease, once admitted, should not be discharged, save for quite exceptional reasons. To improve the patient’s condition a little and then discharge him is almost completely to negative the purpose for which the hospital was instituted.

Such hospitals should preferably be within, or in close proximity to, the area they serve. It is very desirable that the patient’s friends should have easy access to him, and that there should be no suggestion of segregation as in a “leper-house.” From this point of view there is much to be said for the inclusion of this type of hospital as part of a general fever hospital. In some districts, on the other hand, it may be convenient to have smaller homes-of-rest, the benefits of which would be soon appreciated.

The provision of hospital accommodation is also needed to meet emergencies in the course of tuberculous illness, e.g. acute exacerbation, pleurisy, hæmoptysis, and the like, occurring in patients who are otherwise maintaining their ground sufficiently well at home. Such accommodation is frequently available in a general hospital in the area. This is perhaps best, as it allows the emergency to be treated as an incident—often of much interest and importance in the training of students—and gives time for exact observation and the determination of the further course to be followed. The same purpose is attained in connexion with acute types of disease, such as meningitis, acute miliary tuberculosis, etc.

Hospitals may also be made serviceable for purposes of temporary treatment and training of more chronic cases. Much can be gained by teaching such patients how to manage themselves. In some six weeks they can learn much that is invaluable.

## TUBERCULOSIS FROM THE STANDPOINT OF PREVENTIVE MEDICINE

**5. Working Colonies.**—Sanatorium results have shown that in a considerable number of cases complete arrest of tuberculosis cannot be attained within the ordinary time limit of sanatorium residence, even when this extends considerably beyond the impossibly short "three months" commonly meted out to tuberculous patients under the National Insurance Act.

Prolonged experience of sanatorium treatment points to the conclusion that, broadly speaking, patients who have progressed favourably are at the end of a given period, say, 6-12 months, either (1) fit to return to their ordinary occupation, or (2) hardly ready for this—that is to say, immediate return to the *ordinary* conditions of home environment and work would involve undue risk of relapse.

Collective statistics from various sanatoria suggest that within four or five years some 50 per cent. of apparently arrested cases have relapsed. In other words, even the extended time limit which experienced sanatoria demanded as essential failed to secure the complete cicatrization of the tuberculous lesion which is necessary, if permanency of arrest is to be attained.

The significance of the Working Colony is apparent at this point. The Colony should be complementary to the sanatorium, affording a simple, inexpensive means for securing, in the cases referred to, the more permanent arrest of the tuberculous process that was impossible without undue expenditure at the sanatorium. The process of effective cicatrization takes a long time. But the later stages of the process may be carried out on *more economic lines* than those required in the earlier period of treatment.

*Selection of cases.*—It is obvious that if the Colony is to afford results of lasting value, the cases admitted must be carefully selected. To attempt to cater for all types of cases is to court disappointment. The ultimate purpose is to complete the cicatricial process locally and remove remaining traces of systemic disability. The essential object of the Colony is to *fix* the cure. Training in various lines of work, valuable as it is from many points of view, is of secondary importance. Training must be subordinate to cure. Its methods must not run counter to the measures essential for the completion of cure. Whatever be the vocational line adopted, the training must always include the unremitting application

of sanatorium principles to workshops and dwellings.

The Colony should, accordingly, be under careful medical surveillance. The medical superintendent must know tuberculosis thoroughly, and be interested in gardening, farming, and industrial work. He has to adapt the particular job to the degree of recovery, and adjust training in the interest of the individual and the common interest of the Colony.

The experience of such Colonies as have been in existence for a sufficient period to render their observations valuable is favourable.

*Village settlements.*—More recently Colonies of a different character have been established, of which the main underlying principle is described as *voluntary segregation*. In one of these, cases at all stages are under treatment—"early," "middle," and "advanced." It is also arranged that entire families should, in some instances, be housed in separate cottages within the settlement. The suggestion that in this way one institution can serve the purpose of three must be received with considerable reservation.

**6. Tuberculosis Care Committee.**—The purpose of the Care Committee is to deal with economic and other domestic conditions which play a large part in the successful management of tuberculosis. The Care Committee has regard to those tuberculous patients and their households whose circumstances call for special consideration. The Committee will advantageously include representatives of the chief agencies which in the given area are likely to be serviceable in assessing and meeting the claims of cases as they are submitted. The information regarding cases will commonly be furnished to the Care Committee by the Tuberculosis Officer or one of the doctors or nurses attached to the Tuberculosis Dispensary. Inquiry into the home conditions of each patient forms part of their regular work (*see* Schedule, p. 390). The information thus applied is discussed by the Committee with a view to suitable action.

While the Care Committee concerns itself primarily with economic rather than medical matters, the visits of the members of the Committee to the homes of patients—a most important feature of the work—are serviceable in reinforcing the instructions of the doctor and nurse. The home conditions are frequently faulty through the inmates' ignorance of domestic economy. A wise and tactful visitor

## TUBERCULOSIS FROM THE STANDPOINT OF PREVENTIVE MEDICINE

can advise and guide in many helpful directions without necessarily giving more material aid.

The purpose of the Care Committee is not to distribute charity but to ensure that neither the tuberculous patient nor the household of a tuberculous patient suffers hardship which can be prevented. The Committee should be in close touch with the Charity Organisation Society and such agencies. It is thereby in a position to do the best for the patient without risk of overlapping.

The efforts of a Care Committee may conveniently take such directions as :

(1) Improvement of accommodation for the patient and his household through better arrangement of the house, or by the removal of one or more persons to other quarters, or by "fitting" to a larger house.

(2) Provision of extra clothing—bed and personal—when necessary.

(3) Provision of extra food supplies or invalid comforts when necessary.

(4) Guidance as to purchase of suitable food and how best to cook it.

(5) Arrangements for household washing.

(6) Selection of suitable work either for the patient or for other members of the household.

(7) Financial assistance to households when the chief wage earner is unfit for work and perhaps in sanatorium or hospital.

(8) Care of the children, especially when the mother is under treatment.

*Selection of suitable occupation for convalescent tuberculous persons.*—The selection of a suitable occupation for a given patient involves much discrimination. It depends (1) on the medical outlook, and (2) on personal qualification and taste. No two cases are quite the same. The ultimate guide should be the doctor, who is best able to decide as to the suitability or otherwise of the proposed work for a given physical state. At the same time, the doctor is not always the best judge of economic and other trade considerations. Hence the advantage of the Tuberculosis Care Committee discussing questions of occupation. In considering a particular case, it is well to have in view various possibilities which may be proposed to the patient, who naturally claims to have the final word.

The following list includes occupations which have been found suitable in actual cases. Whatever the selected occupation, it is essential that the working accommodation and other conditions should be satisfactory.

### EMPLOYMENTS SUITABLE FOR THE TUBERCULOUS

#### FOR MEN

Basket-makers  
Bath-chairmen  
Canvassers  
Caretakers (if accommodation is satisfactory)  
Carpenters  
Chauffeurs (private, taxi, motor-bus)  
Coachmen  
Collectors (rents, debts, etc.)  
Commissionaires  
Conductors (bus, tramway-car, etc.)  
Drivers (bus, cab, van)  
Farm labourers  
Fishermen (special departments)  
Foresters  
Gamekeepers  
Gardeners (private, market) (*not glass-house work*)  
General labourers (except very dusty jobs)  
Hawkers  
Insurance and commission agents  
Joiners  
Lodge porters  
Motor cleaners  
Painters and decorators  
Park attendants and rangers  
Policemen (if already in the service)  
Porters (light)  
Postmen (if already in the service)  
Sanatorium employees  
Sandwich men  
Ship stewards (if accommodation is good)  
Station-bookstall attendants  
Ticket-collectors  
Timekeepers  
Travellers  
Watchmen  
Window-cleaners  
Woodcarvers  
Woodmen  
Wood-road layers

#### FOR BOYS

(Unless well enough to be apprenticed to a healthy trade)

Errand boys  
Golf caddies  
Messenger boys  
News boys  
Telegraph boys (if already in the service)  
Van boys

#### FOR WOMEN

Button-hole makers  
Caretakers (if accommodation satisfactory)  
Cashiers (if airy premises)  
Charwomen (under good conditions)  
Cork sorters  
Dressmakers  
Farm workers (except in dairy)  
Flower sellers  
French polishers  
Gardeners (private, market, etc.)  
Hop pickers

## TYPHOID AND PARATYPHOID FEVERS

Housemaid work (in easy place, not in charge of children, food, etc.)  
 Ironers, folders, and menders (in laundry)  
 Lace makers  
 Leather workers  
 Message girls  
 Milliners  
 Needleworkers (embroidery, etc.)  
 Net-makers  
 Pea-pickers  
 Poultry farmers  
 Sanatorium servants  
 Secretaries (skilled and unskilled)  
 Shop assistants (in airy premises)  
 Teachers in open-air schools  
 Umbrella makers  
 Waistcoat makers  
 And such occupations suggested for men as may be suitable.

R. W. PHILIP.

**TUBERCULOSIS, MILIARY** (*see* TUBERCULOSIS, ACUTE GENERAL).

**TUBERCULOSIS, PULMONARY** (*see* PULMONARY TUBERCULOSIS).

**TUBERCULOSIS VERRUCOSA** (*see* SKIN, TUBERCULOSIS OF).

**TUBERCULOUS ARTHRITIS** (*see* ARTHRITIS, TUBERCULOUS).

**TUBERCULOUS MENINGITIS** (*see* MENINGITIS).

**TUBERCULOUS PERITONITIS** (*see* PERITONITIS, TUBERCULOUS).

**TUMOURS** (*see* under individual organs).

**TURBINAL BONE, HYPERTROPHY OF** (*see* NASO-PHARYNGEAL OBSTRUCTION).

**TURPENTINE POISONING** (*see* POISONS AND POISONING).

**TYMPANIC MEMBRANE, RUPTURE OF** (*see* OTITIS MEDIA).

**TYPHOID AND PARATYPHOID FEVERS.**—Fever characterized by an eruption of rose spots, intestinal lesions, and enlargement of the spleen, and due to infection with the typhoid bacillus, or with one of the varieties of *B. paratyphosus*. (PLATE 5, Figs. 6-9, Vol. I, facing p. 148.)

**TYPHOID FEVER** (*syn.* Enteric Fever)

**Etiology.**—Transmission of infection is almost entirely effected by the faeces and urine excreted either by the typhoid patient or by typhoid carriers. Apart from the so-called "healthy carriers," it has been calculated that

from 2 to 5 per cent. of all attacked with typhoid fever become chronic carriers, but whether the proportion is so high as this is very doubtful. Such persons, most of whom belong to the female sex, continue to excrete typhoid bacilli for many months, and sometimes as long as thirty to fifty years after an attack of typhoid fever. In many carriers the site of multiplication is probably the gall-bladder. Carriers usually give rise to sporadic cases only, but may be the cause of an epidemic owing to their having contaminated a milk supply.

The disease may be spread by direct contagion, especially in the case of the patient's attendants, whose hands become contaminated by the faeces and urine, the virus being subsequently conveyed to the mouth before they have been properly cleansed. Indirect transmission of infection, however, is much more frequent, and is due to contamination of water, milk, oysters, shellfish, and other articles of food by typhoid bacilli. Contamination of the water supply is by far the most important factor in the spread of the disease. It is effected through the infected excreta obtaining direct access to it or reaching it by gradual infiltration through the soil.

Milk may be infected by a typhoid carrier acting as milkman, or by contaminated water being added to it accidentally or for adulteration, or being used to wash the cans.

The consumption of oysters has on several occasions caused epidemics of typhoid fever owing to the oyster-beds having been situated close to sewage effluents, or to subsequent contamination in the market or elsewhere.

Uncooked vegetables, especially celery, radishes, and watercress, may also harbour typhoid bacilli which have been derived either from contamination of the soil with sewage or human manure, or from the water used to wash them.

An important part in the dissemination of typhoid fever is played by the common house-fly which, after settling on the excreta of typhoid patients or carriers, conveys the bacilli to articles of human consumption. This mode of food contamination is most likely to occur in military camps, as was well shown in the Spanish-American and South African wars, and among the poorer classes of the civil population, whose means of disposal of faeces and urine are inadequate. The inability of the typhoid bacillus to resist desiccation makes it improbable that dust, air, and fomites have much, if any, power to disseminate the disease.

**Predisposing causes.** *Age.*—Typhoid fever

## TYPHOID AND PARATYPHOID FEVERS

may occur at any age, but is essentially a disease of adolescence and early adult life.

*Sex.*—Males are somewhat more frequently attacked than females, being probably more exposed to infection.

*Season.*—The disease is most prevalent in the autumn, and least so in the spring.

*Geographical distribution.*—Typhoid fever has a world-wide distribution, but is most prevalent and most severe in temperate climates. It is endemic in most of the large towns in the United Kingdom, but within recent years there has been a considerable decrease in its prevalence. Newcomers to a town or district in which the disease is endemic are more liable to contract it than the native population.

*Occupation.*—The disease is apt to be prevalent among soldiers in campaign unless they are protected by preventive inoculation, as is shown by the high morbidity in the Spanish-American and Boer wars, and the much lower incidence in the Great War, when inoculation was carried out on an extensive scale in all the belligerent armies. Doctors, nurses, and other members of a hospital staff occasionally contract the disease.

*Immunity.*—One attack of typhoid fever usually confers immunity for the rest of life. Second attacks are as rare as in scarlet fever. They appear to be more common in patients whose original attack has been contracted in some other part of the world.

*Pathology.*—The typhoid bacilli gain entrance by the alimentary tract, where they first attack the lymphoid tissue of the intestinal wall, and then pass by the lymphatics to the regional lymph-glands. They then travel up the thoracic duct and enter the blood, by which they are carried to the spleen, bone-marrow, kidneys, liver, gall-bladder, and elsewhere. Typhoid fever is thus not a true septicaemia but an infection of the lymphoid tissue attended with a secondary bacteraemia. The presence of bacilli in the blood merely represents an overflow from the lymphatic system; so far from multiplying in the blood they are destroyed there in large numbers, and thus are set free the endotoxins which give rise to the symptoms. The bacilli are most numerous in the blood during the first two weeks; as the disease declines their number diminishes, until they disappear when the temperature becomes normal. A relapse indicates reinvasion of the blood by the bacilli, followed by their destruction and the liberation of their endotoxins. It is possible that

in some cases this reinvasion is by a different strain of the organism which has remained dormant throughout the original attack.

*Morbid anatomy.*—The characteristic morbid process in typhoid fever is inflammation and ulceration of Peyer's patches and the solitary glands of the *small intestine*. The lesions usually predominate in the lower end of the ileum and vermiform appendix, and are often confined to this region; but sometimes the lymphoid tissue throughout the ileum and the large intestine even to the rectum is affected. Lesions confined to the large intestine have been known to occur. In rare cases the jejunum and even the duodenum may be involved.

During the first week of the disease the solitary glands and agminated follicles of the intestinal mucosa become swollen and hyperaemic. The process may abort in the initial stage, especially in children, but as a rule ulceration of the lymphoid tissue commences in the course of the second week and gradually increases in depth, usually extending down to the muscularis mucosae. In the third and fourth weeks a greyish-yellow or brownish slough forms and separates.

A typical typhoid ulcer developing in a Peyer's patch is oval in shape, its long axis corresponding to the long axis of the intestine; its base is smooth and its edges are thin and undermined. When only a portion of a Peyer's patch is involved, the outline of the ulcer is irregular, while the ulcers occurring in the solitary follicles are small and round. The lesions are usually most evident in the neighbourhood of the ileo-caecal valve and become progressively less marked above this region. The ulceration may extend through the intestinal wall, giving rise to perforation as the slough separates; but in most cases healing takes place by granulation and epithelial formation in the course of the fourth or fifth week. Cicatricial stenosis of the bowel does not occur, but a punctiform pigmentation may develop at the site of the ulcers and, persisting for many years, allow a retrospective diagnosis of typhoid fever. In recrudescences and relapses the process of inflammation, necrosis, and repair is repeated in the lymphoid tissue hitherto immune, and sometimes in that previously attacked.

Enlargement of the mesenteric glands is a constant feature, and may even be met with in those rare cases which show no obvious naked-eye changes in the intestinal mucosa. The glands corresponding to the affected part

## TYPHOID AND PARATYPHOID FEVERS

of the intestine are chiefly involved, but more or less general adenopathy, in which not only the abdominal but also the inguinal bronchial and cervical glands take part, is usually present. The inflammatory enlargement is considerable, the glands sometimes reaching the size of a small plum. Necrosis may take place and, less frequently, abscess-formation. In favourable cases the glands diminish in size in the course of the third week and gradually return to their normal condition; in rare instances the lesions terminate in sclerosis, caseation, or calcification.

**Spleen.**—Enlargement of the spleen is early and constant, and is more marked than in any other acute infectious disease prevalent in the British Isles. Its consistence at first is firm, but later soft and diffuent. Infarcts are sometimes present, but rare.

**Liver.**—The liver may show accumulations of lymphoid cells (lymphomata), and occasionally small areas of focal necrosis. Abscesses, solitary or multiple, are rare.

The **gall-bladder** is more frequently affected than the liver itself, being very liable to catarrhal inflammation due to the large number of typhoid bacilli which reach it by the portal vein, hepatic artery, or common bile-duct. A further degree of inflammation causes ulcerative cholecystitis which may end in perforation.

**Heart.**—The principal lesions are found in the myocardium, which is pale and flabby, and shows yellowish-brown coloration or definite mottling on section. There may be no naked-eye changes, but in two-thirds of the fatal cases microscopical examination reveals parenchymatous or interstitial myocarditis and lesions of the small vessels of the myocardium (Hoffmann). Endocarditis is rare; in a few cases typhoid bacilli have been isolated from vegetations on the valves. Pericarditis also is rare, and usually a necropsy surprise. It may be dry, or accompanied by effusion which is usually purulent.

The **larynx** is not infrequently affected, the lesions ranging from mild catarrhal laryngitis to complete necrosis. Ulceration may be confined to the epiglottis, but more frequently starts in the posterior wall of the larynx, causing perichondritis of the cricoid, and less frequently of the arytenoid and thyroid cartilages. The **trachea** and **bronchi** may be similarly affected.

**Lungs.**—Hypostatic pneumonia is not uncommonly found in patients who have died

at the height of the disease or after severe and prolonged attacks. Lobar pneumonia due to the typhoid bacillus or the pneumococcus or to a mixed infection is occasionally present. Gangrene of the lung may be a sequel of a lobar pneumonia or originate from deglutition broncho-pneumonia. Hæmorrhagic infarction may be caused by a thrombus in the right auricle or in a branch of the pulmonary artery. Pleurisy, either serous or purulent, is rare. Typhoid bacilli may be found in the fluid in either case.

**Nervous system.**—Gross changes in the brain, such as hæmorrhage, softening, and abscess, are rare. Cerebral oedema is frequent, and is said by Hoffmann to occur in one-third of all cases. Meningitis is uncommon. It may be serous or, less frequently, suppurative. The cerebro-spinal fluid contains polymorphonuclear leucocytes and typhoid bacilli; there are also agglutinins, which are less abundant than in the blood-serum. The meningitis may be secondary to other suppurative lesions such as otitis media or parotitis, or may occur independently. Several cases of meningitis due to the typhoid bacillus but without any intestinal lesions have been reported. Autopsies on well-marked cases of myelitis show diffuse parenchymatous, vascular and interstitial lesions. Peripheral neuritis, due either to the typhoid bacillus or to its toxins, has been found in several cases, unassociated with clinical symptoms.

**Muscles.**—There are three varieties of muscular lesions: (1) The granular and waxy degeneration of the striated substance described by Zenker and others, occurring in the acute stage. The recti abdominis, psoas, pectorals, and leg muscles are chiefly affected, and may show hæmorrhages, rupture, or abscesses as a result of this condition. (2) Hypertrophic myopathy, usually due to vascular lesions, especially phlebitis. (3) Progressive pseudo-hypertrophic or atrophic myopathy.

**Kidneys.**—When death occurs early in the disease the kidneys are usually enlarged and hyperæmic. Later they tend to become pale and to diminish in size. Actual nephritis of the hæmorrhagic parenchymatous type is less frequent. Infarcts and abscesses are occasionally present, and lymphomatous nodules are sometimes found in the cortex. Apart from catarrhal cystitis, which is not uncommon, the rest of the urinary tract is rarely affected.

Orchitis and epididymitis are exceedingly infrequent.

## TYPHOID AND PARATYPHOID FEVERS

**Bones.**—Periostitis and less frequently osteomyelitis may affect the long bones, especially the tibia, ribs, and clavicle. The inflammation may be due to the typhoid bacillus alone or to its association with other organisms, such as the streptococcus, staphylococcus, *B. coli*, or proteus. The typhoid bacillus may remain quiescent in the bone-marrow for months and even years after an attack of typhoid fever, and only give rise to osteomyelitis when the local resistance has been lowered by any cause, such as trauma. The condition of "typhoid spine" calls for special mention. It consists of a spondylitis and perispondylitis. X-rays show changes in the intervertebral discs, thickening of the perivertebral tissue and vertebral periosteum, and lesions in the bodies of the vertebrae.

**Blood.**—A moderate degree of anæmia, such as occurs in most severe infectious diseases, is the rule. The average loss in the red cells is 1,000,000 per c.mm. (Thayer). The fall in the hæmoglobin is relatively greater than in the red cells, as is usual in secondary anæmias. The anæmia is increased by intestinal hæmorrhage, and may continue for some weeks after the temperature has become normal. Apart from the nucleated forms sometimes seen after intestinal hæmorrhage, the red cells rarely undergo any qualitative changes. The changes in the leucocytes are of greater interest and importance. Except in the presence of inflammatory complications such as bronchopneumonia, parotitis, peritonitis, diarrhoea, and nephritis, and in intestinal hæmorrhage, which are all associated with leucocytosis, there is no increase in the total number of leucocytes. As a rule there is a leucopenia which bears a direct relation to the severity of the attack. The total number of leucocytes is usually below 6,000, and may fall to 1,000 or even lower (Curschmann). Though the above-named complications are usually attended by leucocytosis, in profound asthenia no leucocytic reaction may occur. On the other hand, leucocytosis is sometimes present without any complication. It is important to avoid making a blood examination directly after the cold-bath treatment, as this gives rise to a leucocytosis in the peripheral vessels. Various changes may take place in the different varieties of leucocytes. In the first week there is a relative increase in the polymorphonuclears and a similar decrease in the large mononuclears and lymphocytes, while there is an almost entire absence of eosinophils. In the

second and third weeks the relative proportions of the polymorphonuclears and large mononuclears and lymphocytes are reversed. The return of the eosinophils immediately precedes the onset of convalescence.

**Symptomatology.**—The *incubation period* of enteric fever is usually about ten days to a fortnight, though under conditions of great fatigue, hunger, or inanition, or as the result of an unusually large dose of the virus, it may be less than a week. On the other hand, a three weeks' incubation is seen occasionally.

**Onset.**—The attack, as a rule, develops gradually. Headache, lassitude, and muscular pains are first complained of, often accompanied by shivering and marked disinclination for exertion. The appetite fails, the tongue becomes coated, and sore throat is often present in the early stage, attended perhaps by some degree of deafness. Slight bronchial cough is usually to be noted, and the patient, though drowsy, complains of inability to get good sleep, his rest being disturbed by worrying dreams, from which he wakes up unrefreshed, with consequent aggravation of the headache. Epistaxis at this stage is not uncommon. Vague, colicky pains in the abdomen are usually experienced about this time, and some disturbance of the bowels, the latter usually in the form of diarrhoea, which, though rarely persisting in mild attacks, is nearly always present for a day or two. During this time the temperature, though perhaps somewhat irregular at first, will have been rising steadily. The patient, if a worker, may succeed in fighting against his symptoms for some days, but eventually, perhaps not until the expiration of a week, comes under medical observation. In a good many instances, however, the attack develops much more rapidly, and in the suddenness of its onset bears a close resemblance to influenza. Intensity of the headache and muscular pains with excessive prostration, often attended with vomiting, are the chief characteristics of such cases, and the patient is forced to give in from the onset.

An ordinary case of enteric fever seen in bed at the end of the first week will probably present the following appearances: The patient is dull and listless, with slightly flushed cheeks, glad to be in bed, and suffering from severe headache. The mind is unimpaired, and he can give a clear account of his symptoms. The tongue is coated with a pasty fur, and the pulse somewhat accelerated. At this stage the

## TYPHOID AND PARATYPHOID FEVERS

pulse-rate is rarely more than 100, often less, though the temperature may be three or four degrees above the normal. The abdomen will probably be somewhat full, and is often slightly tender, especially in the right iliac region. The bowels are apt to be rather loose, but the stools will not yet have assumed the typical "pea-soup" appearance so characteristic of typhoid. In mild attacks constipation is more often the rule, and diarrhoea, except for a day or two at the onset, conspicuous by its absence. Some increase in the area of splenic dullness can usually be made out on careful percussion, and the lower border of the organ may be felt beneath the ribs on full inspiration. The lower abdominal reflex is usually absent; while the upper, though often present, is apt to be less brisk than it should be. About this time a few spots, rose-coloured, and slightly raised, may appear on the surface of the abdomen, flanks, and lower part of the chest. The urine may contain a trace of albumin, and usually gives the diazo reaction.

During the course of the *second and third weeks* the symptoms undergo aggravation; the patient becomes more severely ill, and presents the aspect commonly associated with typhoid fever. There is marked pallor of the face, surmounted by a hectic flush, and progressive loss of flesh, proportionate to the height and duration of the fever. The mental faculties become impaired as time goes on. For some days, though he may have shown a tendency to wander at night time, the patient is able to pull himself together and answer rationally when spoken to, but such ability disappears after a time, and the nocturnal wandering gives place to continuous, and usually muttering delirium. Occasionally, the delirium is violent in character, especially in strong men, and calls for the services of an attendant. The temperature during the second and third week tends to assume the remittent type, the morning record being  $1\frac{1}{2}^{\circ}$ - $2^{\circ}$  lower than the evening. At this period of the illness the patient's temperature, as shown by four-hourly records, remains fairly constant, averaging from  $101^{\circ}$  to  $103^{\circ}$  F., according to the severity of the case. The pulse-rate during the acme of the fever, which is usually reached between the middle of the third and fourth weeks, will probably have risen to 120, and in toxæmic cases one of 130 is not uncommon. The pulse is of low tension, with a tendency to undulation, and may be definitely dicrotic. The tongue becomes more coated and tends to

become dry and brownish. In severe cases, however, especially towards the tip and edges, it is apt to present a red and glazed appearance. It is often fissured, and sordes is apt to collect on the teeth and gums. Bronchial catarrh is usually pronounced, together with a corresponding increase in the rapidity of respiration, and scattered rhonchi may be heard on auscultation.

The abdomen may have become somewhat more tumid at this period of the illness, but with proper dieting no great degree of distension should be witnessed. Considerable tenderness may be present, but if of slight degree the patient may not be in a condition to recognize it.

In an attack of such severity as here described diarrhoea will probably be present, and the stools during the course of the second week will have taken on the typical "pea-soup" character. From three to six actions of the bowels in the twenty-four hours are not uncommon, but much will depend on the dietary. During the third and fourth weeks sloughs, occasionally tinged with blood, may be expected to appear in the stools; it is during this stage of the attack, when the sloughs are separating, that severe hæmorrhage and perforation of the bowel are likely to occur and gravely affect the prognosis. Fresh spots, coming out in crops and lasting three or four days, may appear at any time in the attack from the sixth or seventh day onwards. They are usually limited to the trunk, and vary in number from half a dozen or so to several hundreds. The urine tends to be scanty and is often albuminous. At any time after the full development of the fever it may present a turbid appearance as the result of bacilluria.

If the case be very severe, muscular tremor affecting also the tongue, subsultus, hypostatic congestion of the lungs, with rapid breathing and cyanosis, retention of urine, loss of control over the evacuations, and stupor may supervene and complete the picture of the so-called "typhoid state," a condition long recognized as the culmination of profound toxæmia. Yet the patient may recover.

Should the case prove fatal from the severity of the fever, death is likely to occur at about this juncture. If, on the other hand, recovery takes place, improvement commonly commences towards the end of the third or at some time during the course of the fourth week, and the first sign is usually a greater fall



## TYPHOID AND PARATYPHOID FEVERS

of temperature in the morning than hitherto. A gradual and progressive fall of the morning temperature goes on until the normal is reached, the pyrexia then becoming intermittent; but it may not be until after several days that the evening records follow suit, and the temperature remains normal throughout the twenty-four hours. Associated with the fall of temperature a general improvement in the patient's condition will usually be noticed. The mind becomes clear during the day, a tendency to wander at night, however, remaining for a few days longer. The tremor, subsultus, and other signs of nervous prostration pass off, and the patient begins to show an interest in his surroundings. The diarrhoea and other abdominal symptoms now abate; the tongue becomes more moist, and gradually cleans, while the appetite returns, and, indeed, often becomes ravenous before the temperature has finally settled.

Coincidentally with the commencement of improvement the urine is secreted in greater quantity, and any albuminuria which may have been present usually clears up. Definite polyuria at this time sometimes occurs, and the skin acts more freely. The length of time it will take a patient to regain his strength will depend upon the amount of flesh he has lost, and his powers of recuperation, the former being chiefly proportionate to the height and duration of the pyrexia, and dependent to some extent on the way in which he has been dieted. It will probably be a fortnight after the subsidence of the fever before he is able to be up, and even then there will be a liability to swelling of the ankles.

The above description may be regarded as applying to a case of rather more than average severity, the fever lasting a full four weeks from the commencement. More cases occur in which the clinical picture, though typical enough, at no time approaches in gravity the condition commonly spoken of as the "typhoid state," but in an adult it is seldom that the duration of the fever is less than three weeks. In children and in persons who have undergone preventive inoculation the attack is often a good deal shorter. A rapid defervescence is apt to occur towards the end of the second week, the illness being over in a fortnight.

In some adults the pyrexia is unduly prolonged, quite apart from relapse, or definite recrudescence, the signs of which are usually sufficiently obvious. In such cases it would seem that the normal defervescence had been

for some reason interrupted, the night temperature being sustained, though the morning remissions may have been quite satisfactory. In this way the pyrexial stage may be prolonged into the fifth or even the sixth week, yet the patient does not seem much the worse for it. The explanation is not very clear, though it is possible that delay in the healing of some of the ulcers and consequent septic absorption may be responsible. In some instances the continuance of slight pyrexia may be due to constipation, in others, perhaps, to inanition, since an increase in the dietary will sometimes prove an effective remedy. Occasionally it appears to be due to too-long detention in bed, since the mere fact of getting the patient up is sometimes followed by its immediate cessation.

Certain symptoms of enteric fever call for more detailed description.

**Temperature.**—It is usual to speak of three stages. (1) The *ingravescent*, or period during which the pyrexia is developing, the temperature mounting daily, step by step, until the maximum level is reached at about the end of a week from the commencement of the illness. In many cases it is less prolonged, a period of four or five days being perhaps more often its duration. (2) The *fastigium*, or developed stage, when the fever is running at its height, and is characterized by a morning remission of  $1^{\circ}$ - $2^{\circ}$ . This usually lasts a fortnight or more, though sometimes less in the milder cases. The average level at which the temperature runs during this stage, as evidenced by four-hourly records, may be regarded as a measure of the general severity of the attack, in the absence of any inflammatory complications. (3) The *defervescent*, or stage of decline, marked by a gradually increasing fall of temperature in the morning, the night record usually at first showing no corresponding improvement. The pyrexia thus displays a notably "spiked" appearance when charted. The period of decline may take about a week before it is complete, though it may not be until after the first three or four days that the night temperature shows any indication of falling.

**Tongue.**—At first the tongue is simply coated with a pasty fur; later it tends to become dry and brownish. During the height of a severe attack it usually presents a somewhat characteristic appearance, its centre being caked with fur with transverse cracks, while its edges and tip have a red and somewhat raw appearance.

## TYPHOID AND PARATYPHOID FEVERS

In some cases the whole surface of the tongue is red, glazed and deeply fissured. Much, however, depends upon the amount of attention given to the mouth by the nurses.

**Stools.**—If a hundred consecutive attacks of enteric fever be observed, irrespective of their severity, constipation will be found to be the rule, and diarrhoea the exception. In severe attacks, on the other hand, looseness of the bowels of some degree is rarely absent, at any rate in the British Isles. The diarrhoea is not likely to be excessive, however, if the diet has been suitable. During the first few days of the fever the bowels are usually irregular, and the motions of variable consistence. It is not until the second week in cases with diarrhoea that the stools begin to assume the characteristic yellow ochre-coloured, pea-soup-like appearance. Their odour is most offensive. During the third week, and later, mucus, shreds of necrotic lymphoid tissue and sloughs, often tinged with blood, are usually to be seen as they become detached from the ulcerated mucous membrane. Afterwards, if recovery ensue, the stools soon resume their normal appearance and consistence.

**Condition of abdomen.**—This varies greatly. The abdomen may be of normal appearance and devoid of tenderness throughout, but is more often somewhat tumid and tender. Much tenderness usually implies extensive, and especially deep, ulceration; in the latter case, owing to irritation of the peritoneal surface of one or more of the ulcers. Extensive ulceration predisposes to distension, but anything approaching meteorism, apart from peritonitis, should never arise with judicious dieting and the efficient administration of antiseptics.

The splenic enlargement, which may have been obvious as early as the end of the first week, rapidly disappears on the commencement of defervescence, and the superficial abdominal reflexes will usually have regained their normal activity.

**Rash.**—This is seen in about 70 per cent. of cases, and is more likely to be absent in children. Though usually confined to the trunk, when copious it may overflow, as it were, on to the proximal end of the limbs, but for spots to appear on the distal end of the legs or arms, still more on the face, is very exceptional. The spots are papular, rose-coloured, and usually, though by no means necessarily, rounded, varying in size from a large pin's head to  $\frac{1}{8}$  in. in diameter. Though very faint at first, and but little raised, they become more

evident by the following day, though they can always be obliterated by pressure with the finger. They come out in crops of from three or four to a dozen or more at any time from the end of the first week onwards. Each spot lasts for three or four days and then gradually fades; it never becomes petechial. The spots are most distinctive in their early phase.

**Types of attack.**—Quite apart from the variation in severity which characterizes different examples of the usual form of enteric fever, cases are occasionally met with which present special peculiarities, and justify their being classed as atypical. We recognize, for instance, a *toxic* form in which the symptoms point to special intensity of the typhoid poison, the usual abdominal indications being overshadowed by the degree of toxæmia. An unduly high temperature, averaging  $103^{\circ}$  to  $104^{\circ}$  F.; severe cardiac depression, evidenced by great rapidity of pulse, with weak systolic impulse, and consequent liability to cyanosis and pulmonary congestion; a hæmorrhagic tendency, as evidenced by bleeding from the gums or elsewhere and, perhaps, by petechiæ, are all indications in this direction. Such cases usually end fatally towards the end of the second week from cardiac failure. Another form of attack, sometimes spoken of as *ataxic*, is met with in which the nervous system appears to be especially affected. Early impairment of the mental faculties, insomnia, delirium, muscular tremor, and general prostration are its special characteristics. Persons of high intellectual capacity, "brainworkers," excessive drinkers, and neurotic children are particularly prone to it. There is also the *fulminant* form, in which the symptoms develop with great intensity and the patient, becoming unconscious, dies after a very few days, probably with hyperpyrexia. Such attacks are rare in these islands, though not infrequent in the tropics. In the so-called *ambulatory* form the patient is so little ill as to be able to pursue his ordinary avocations; it may be only by reason of some accident, such as hæmorrhage from the bowels or a sudden perforation, that the presence of the disease is detected. Cases are sometimes seen in which, though the fever may run its normal course at first, there comes a time, probably towards the end of the second week, when the attack stops unexpectedly and convalescence is quickly established. Such cases have been designated *abortive*, or *fourteen-day fever*, and are not very uncommon in children. An *apyrexial* form is described in which

## TYPHOID AND PARATYPHOID FEVERS

all the cardinal signs of the disease are present except pyrexia. It is said that such attacks may be severe but usually terminate favourably.

**Complications and sequelæ.**—The enteric-fever patient is surrounded by many dangers, for as well as the risks from heart failure, hypostatic pneumonia and congestive bronchitis, conditions directly associated with toxæmia, and more likely to supervene in severe attacks, there are those directly dependent upon the intestinal ulceration, viz. hæmorrhage and perforation of the bowel, which are a constant menace in the later stage of the fever, however mild. He is also liable to various inflammatory complications representing more or less remote manifestations of the activities of the typhoid organism, and to others of a septic nature which are prone to arise in debilitated subjects.

**Cardiac failure.**—More deaths are directly attributable to this than to any other cause. Although the risk of its occurrence is undoubtedly proportionate to the degree of pyrexia, it occasionally supervenes in cases where the temperature has been running quite a moderate course and where there has been no special reason to anticipate its occurrence. Alcoholics, fat people, and those above middle age are especially prone to cardiac failure. Enfeeblement of the heart is evidenced by diminished intensity of the first sound, a rapid, thready, and especially an irregular pulse, and the early supervention of hypostatic pulmonary congestion, attended with increased rapidity of respiration and a tendency to cyanosis. Collapse often sets in very suddenly, and death is not long delayed.

**Relapse** occurs with varying frequency; a fair average is from 10 to 15 per cent. The interval between the subsidence of the original pyrexia and the relapse is usually six to ten days; it is rarely more than a fortnight. Often, however, there is no apyrexial interval, the temperature gradually rising again before defervescence is complete. It is usual to speak of this as a "recrudescence" rather than as a "relapse," but there is no essential difference between them.

Relapses are commonly less severe and less protracted than the primary attack, though presenting the same characters. The temperature rises daily step by step, taking three or four days, as a rule, to reach its full development; the pulse-rate increases *pari passu* with the pyrexia, and fresh spots are apt to come

out about the third to the fifth day of the relapse. The tongue again becomes coated, but rarely shows the dry, cracked, or even glazed appearance so characteristic of typhoid fever. The abdominal reflex commonly disappears; some degree of enlargement of the spleen can often be made out, and the urine again gives the diazo reaction. It is an interesting fact that although the temperature in relapse may reach the same height as in the primary fever, and the pulse be as rapid or even faster, the patient is rarely affected to the same degree. He does not seem so ill, and delirium is usually absent. The stools may again become liquid, and the abdomen show a somewhat tumid appearance; yet, although fresh intestinal ulceration usually accompanies a relapse, neither serious diarrhœa nor hæmorrhage is often seen, while perforation of the bowel is most exceptional. The duration of a relapse seldom exceeds ten days or a fortnight, but it may be very much longer.

**Intestinal hæmorrhage** occurs in about 8 to 10 per cent. of cases. Apart from an occasional streak of blood in the stools, or the presence of mucus showing a reddish tinge (both of which are congestive in origin and liable to occur at an earlier stage), definite hæmorrhage from the bowels is not likely to be met with before the end of the second week, for it is associated with the ulceration and the separation of the sloughs. Like perforation, hæmorrhage is more common in the third and fourth weeks, and is more prone to arise in cases where diarrhœa is a prominent feature. Females are especially liable during the menstrual periods. The amount of blood lost varies enormously. Should the amount be small, the presence of some blood in the motions, with, perhaps, a slight lowering of the pulse-tension, may be the only indications; but when the loss of blood is considerable, amounting perhaps to a pint or more, signs of collapse will be more or less evident. Pallor, restlessness, a drop in temperature of several degrees, a rapid feeble pulse, together with a complete absence of abdominal pain, tenderness, or rigidity, may then be expected. The patient usually complains of dryness of the mouth, and may suffer much from thirst, the degree of which is usually proportionate to the amount of hæmorrhage. The fact that it is advisable to restrict within the lowest possible limits the amount of fluid the patient takes, and for which he craves to quench his thirst, is unfortunately calculated to aggravate his suffer-

## TYPHOID AND PARATYPHOID FEVERS

ing. Should the hæmorrhage be moderate in amount, and not recur, its effect need not be prejudicial. Indeed, improvement often dates from the occurrence of the bleeding. Although the actual effusion of blood into the bowel may soon cease, the stools usually contain some blood for several days afterwards. That passed in the motions will at first be bright in colour, with numerous clots; it gradually becomes less in amount, and more intimately mixed with fecal matter, until the motions, as a result of their admixture with altered blood, present a tar-like appearance and are very offensive. In some instances the bleeding, though small in quantity, recurs so frequently as to be practically continuous. These "leaking" cases are of very unfavourable augury, the patient's strength being gradually reduced by the continual drain. In exceptional cases a typhoid hæmorrhage may be so sudden in onset and so overwhelming in quantity as to prove directly fatal, pure blood to the extent of several pints being passed without previous warning. The face becomes blanched and the pulse rapidly imperceptible. Intense restlessness, sweating, faintness, and wandering delirium set in, and the patient, with subnormal temperature, dies collapsed from loss of blood within an hour or two of the first appearance of the hæmorrhage.

**Perforation of the bowel.**—This, the most dreaded of all complications, is met with in from 2 to 4 per cent. of attacks. It most often occurs in cases in which the diarrhœa has been excessive, the time of onset coinciding with that at which the sloughs are separating from the intestinal ulcers. This is from the commencement of the third week onwards. Perforation is not very often met with after the temperature has reached the normal, nor indeed after the commencement of definite defervescence. It is often preceded, and occasionally accompanied, by hæmorrhage. Children are almost exempt, and it is not very common in patients over 40.

The most characteristic symptom of perforation is the sudden occurrence of acute abdominal pain. This may be referred to any part of the abdomen, but more often, perhaps, to the neighbourhood of the umbilicus or the ileo-cæcal region. It is accompanied by exquisite tenderness at some point in the abdomen and defensive rigidity of the abdominal muscles, the effects of local peritonitis. The pulse-rate is quickened, and the facial expression denotes anxiety. The latter, however, like the

pain and tenderness, may be but slightly marked in the presence of profound toxæmia. Vomiting, shivering, or an actual rigor may occur at the moment of perforation, but the last is exceptional.

The further progress of the case is indicative of general peritonitis. Vomiting and retching continue and tend to become intractable. The area of tenderness rapidly spreads so as to involve the whole of the abdomen, which is rigid and immobile, the breathing being entirely thoracic. Meteorism soon develops, although the abdominal wall, as the immediate result of perforation, may previously have been somewhat retracted. Owing to the escape of gas into the peritoneal cavity, the hepatic dullness becomes obliterated, and the gradual supervention of more or less dullness in the flanks denotes the presence of some ascites. The pulse becomes more rapid, feeble and threadlike, and ultimately imperceptible. As the collapse increases, the patient's expression becomes more anxious, the features pinched and drawn, with sunken eyes and pallid or greyish complexion. The constant retching occasions much distress, the skin is bathed in sweat, and the urine often completely suppressed.

The temperature, which may have fallen to normal, or even lower, on the occurrence of perforation, usually rises several degrees with the onset of peritonitis. The mind, though remaining inordinately clear throughout, is apt to wander just before the end, which, in the absence of early operation, is rarely delayed more than forty-eight hours from the time of perforation.

Although the signs of perforation are usually fairly evident, its occurrence is sometimes very insidious, especially if the patient be very ill as the result of severe toxæmia. In these circumstances many of the classical symptoms on which we rely for a diagnosis of perforation may be either conspicuously absent or inappreciable, and its occurrence be unsuspected. In our experience the presence of abdominal rigidity, the facies, a running pulse, and loss of liver-dullness are the signs least likely to fail one. As regards the latter symptom, care must be taken not to mistake the partial obliteration of hepatic dullness, due to gaseous distension of the colon, for its comparatively sudden and complete extinction resulting from the admission of air into the peritoneal cavity. The possibility of error in this direction serves but to emphasize the paramount importance

## TYPHOID AND PARATYPHOID FEVERS

of never neglecting to make a careful daily examination of the abdomen in every case of enteric fever. Although a large increase in the number of the polymorphonuclear cells in the blood is usually present in peritonitis, this phenomenon may be due to other causes. A leucocyte count, therefore, is not likely to be of much use in the diagnosis of perforation.

**Bronchitis.**—The bronchial affection is occasionally so pronounced as to justify its being recorded as a definite complication. It is likely to occur in an aggravated form in persons of middle age and over, in drinkers, and in those previously subject to bronchitis. It is usually most pronounced during the height of the fever, yet in many severe attacks, even though the air-passages may have hitherto remained unusually clear, the hypostatic engorgement of the lungs resulting from cardiac enfeeblement, which is apt to supervene at a later stage, is always associated with a good deal of bronchial congestion.

**Pneumonia.**—The difficulty often encountered in diagnosing pneumonia in its early stage from typhoid fever is fully recognized, but it must not be forgotten that pneumonia is occasionally found as a complication of the latter disease, the two affections coexisting. The pneumonia, as a rule, comes on in the early stage of the fever, and it may be only after the occurrence of the pneumonic crisis that the true nature of the illness is revealed. The fact that the temperature remains more or less elevated after the critical fall, the presence of spots, the condition of the abdomen, and a positive Widal reaction are likely to be the most helpful indications. The pneumonia, as a rule, runs its usual course, though the combined effect of the two diseases is likely to aggravate the patient's danger.

**Pleurisy.**—This is not a frequent complication, but appears to be more common in females. Its onset is apt to be insidious, though some pain in the side is commonly complained of. There is usually some effusion, which, however, tends to clear up rapidly, and but rarely goes on to empyema.

**Phlebitis.**—Thrombosis of one or more of the large veins of the leg, viz. the femoral, popliteal, or internal saphenous, is a complication met with in 2 or 3 per cent. of cases, though in tropical countries, such as India or South Africa, the incidence appears to be a good deal higher. It comes on, as a rule, in early convalescence, sometimes before the temperature has settled to normal, and seems to be

connected with an increase in the normal calcium content of the blood. The suggestion, however, that the liability to thrombosis is dependent on the long-continued use of an exclusively milk diet is hardly convincing.

The onset of phlebitis may be suspected from the occurrence of pain and tenderness in the front of the thigh towards the apex of Scarpa's triangle, the midline of the popliteal space, or in the muscles of the calf below it. The temperature is usually raised several degrees, and occasionally shivering is noted at the time of onset. The thrombosed vein can often be felt as a tender, cord-like structure lying deep in the tissues, and any doubt as to the nature of the condition will be at once dispelled by the super-vention of œdema of the limb below the point affected. The left leg suffers more often than the right. The inflammation tends to spread along the course of the vein, and, if the femoral be involved, may even reach the common iliac. In this case the other leg may be affected by extension. More often, however, when the other leg is involved as well, the thrombosis arises independently. After the inflammation has subsided the affected vein can still be detected as a thickened cord on palpation. The swelling of the limb may take many weeks, or even months, to subside entirely, and is liable to recur on walking. Occasionally the œdema becomes permanent, and the patient's capacity for locomotion may be seriously restricted owing to the aggravation of the swelling, and consequent pain, after walking any distance.

**Peritonitis unconnected with perforation.**—This is usually local, and not necessarily serious. It is for the most part due to extension of inflammation through the bowel wall from one or more of the ulcers until the peritoneal surface is reached. The tenderness is usually localized, though pain may be referred to other parts. It is attended with some rigidity of the abdominal muscles and, perhaps, some increase of pyrexia. Peritonitis is occasionally due to suppuration of a mesenteric gland, and, even more rarely, to perforation of an ulcerated gall-bladder; in the latter case it is very serious and resembles that due to intestinal perforation, for which, indeed, it is usually mistaken.

**Parotitis** is met with occasionally, usually during the height of the fever, and seems to depend upon oral sepsis. It is rarely bilateral, but suppuration is not uncommon, peculiarities which serve to distinguish it from mumps.

## TYPHOID AND PARATYPHOID FEVERS

**Cholecystitis** is seen as an occasional complication, the inflammation being dependent upon invasion of the gall-bladder by bacilli which probably gain access to it through the bile-ducts. It sometimes arises during the fever itself; at other times after a prolonged interval, which is explained by the fact that bacilli may persist in the gall-bladder for many years after the attack. Ulceration of the gall-bladder is occasionally found post mortem, and is apt to be associated with gall-stones. One of the ulcers may perforate and set up fatal peritonitis.

**Nephritis** is sometimes met with as the most striking manifestation of the typhoid poison. Indeed, it is apt to overshadow the more characteristic symptoms of the disease to such an extent that it may be only after an examination of the blood or the detection of bacilli in the urine that the true nature of the illness is revealed. The attack, however, usually terminates favourably.

**Cystitis** develops occasionally, usually in the later stage of the attack. It is rarely severe, and the urine, as a rule, remains acid or neutral. Bacilluria is said to be present in about one-fifth of cases of typhoid fever; in a good many the *Bacillus coli* is the infecting organism. The presence of bacilli in the urine does not necessarily imply the occurrence of cystitis, though it is often present, and not all cases of cystitis are associated with bacilluria.

**Cancerum oris** is occasionally seen in very debilitated subjects. Neglect of the daily toilet of the mouth on the nurse's part is a predisposing factor.

**Meningism**, as a complication of a severe attack, is sometimes seen in children. Though simulating meningitis very closely, it is often much improved by lumbar puncture, and recovery is common. A true meningitis is much less frequent.

**Rigors** are apt to occur in the course of enteric fever, often without any ascertainable cause. The rigor comes on suddenly, the temperature rising to 104° F. or more, and the patient is very ill. The shivering soon ceases, perspiration follows, and the temperature falls again in a few hours to its former level. The rigor, however, may recur several times at intervals of a few days.

**Bedsore**s are very prone to arise in enteric fever, and call for very careful and efficient nursing.

Among the complications which are liable

to arise *during convalescence* the following are deserving of mention.

**Periostitis** of the shaft of one of the long bones, the tibia, ribs, femur, and clavicle being most often affected. A tender swelling, with redness of the skin, appears at some point on the surface of the bone, accompanied by lancinating pains and some pyrexia. Suppuration of the swelling occurs in the majority of cases.

**Dementia** occasionally supervenes in early convalescence. It is practically confined to cases in which delirium has been a prominent feature of the attack, the subsequent mental disorder being engrafted upon it. Melancholia and suspicion are usually pronounced, the patient often refusing to speak or eat by virtue of some fixed delusion. On the other hand, constant chattering, whining, or the uttering of piercing cries may characterize other cases. After a few weeks the condition passes off, recovery being almost invariable.

**Peripheral neuritis** is an occasional sequel, the tibial, anterior crural, and intercostal nerves being those most often affected. The affection is apt to be very troublesome and persistent, the painful neuralgia of the earlier stage being usually followed by wasting. The so-called "**typhoid spine**" is met with, though very rarely, in typhoid convalescents, young adult males being commonly its victims. Though possibly a neurosis pure and simple in some cases, it would appear to be due more often to definite inflammation of the ligaments of the spinal column, possibly of a rheumatic nature in certain instances. Most patients complain of intense tenderness on pressure over the dorsolumbar region, with hyperæsthesia of the skin, while the slightest attempt to flex the spine in any direction gives rise to excruciating pain. The affection is most intractable, recovery usually being a matter of several months.

**Multiple abscesses**, localized usually in the subcutaneous tissue, are sometimes met with in patients, generally children, who have been much reduced by long continuance of the fever. Though pyæmic in nature, the infection is of low virulence, and rarely proves fatal if the collections are promptly evacuated as they arise. The patient, however, is commonly reduced to the lowest pitch of emaciation before improvement commences.

**Diagnosis.**—Identification of enteric fever in the incipient stage is admittedly not easy, but even by the end of the first week, or the

## TYPHOID AND PARATYPHOID FEVERS

beginning of the second, a time when the indications might be expected to be fairly obvious, its recognition may be very difficult, especially in cases where definite evidence of abdominal inflammation is wanting.

Much assistance may be looked for from the bacteriologist owing to recent improvements in laboratory technique, but even at this stage the evidence yielded by bacteriological examination may be quite inconclusive, and it may only be possible to arrive at a diagnosis by a process of exclusion. Speaking generally, should a careful physical examination of the patient fail to reveal the presence of inflammation of some organ or other structure, or of any special dyscrasia sufficient to explain it, a continued pyrexia of a week's standing is more likely to be due to typhoid infection than to anything else, at any rate, in the British Isles. The pyrexia of influenza rarely lasts so long, and typhus is not likely to be met with, though both diseases bear a resemblance to many cases of typhoid in showing no sign of localized inflammation.

Of symptoms which point to a diagnosis of typhoid, the presence of spots may be regarded as practically conclusive, even though some may be anything but typical, while definite enlargement of the spleen, absence of abdominal reflex, and the slow pulse-rate are highly suggestive.

The suspicion is strengthened if the illness is stated to have developed gradually and to have been attended with persistent frontal headache; while slight bronchial cough, restless nights, sleep being disturbed by harrowing dreams, epistaxis, the occurrence of transient diarrhoea and of colicky pains in the abdomen (apart from the taking of aperient medicine) are confirmatory. Slight sore throat and some degree of deafness are often complained of early in the illness.

At a somewhat later stage the remittent type of temperature, the development of tenderness with slight tumidity of the abdomen and typical pea-soup stools, if present, will materially assist the diagnosis, though it should be remembered that in mild and even moderately severe attacks constipation is more likely to be present throughout. A retracted abdomen, so common in meningitis, and occasionally present in tuberculous peritonitis, is never seen in typhoid fever.

**Laboratory diagnosis.**—The most reliable criterion by far is the detection of the typhoid bacillus in the blood, stools, urine, or rose

spots, the examination of which, however, demands the services of a skilled bacteriologist. Bacilli can rarely be recovered from the blood before the fifth day of the fever, but from this date until about the end of the second week a positive result may be looked for. For the purposes of a blood-culture at least 5 c.c., or preferably 10 c.c. of blood, should be taken from one of the large veins at the bend of the elbow with strict aseptic precautions.

Bacteriological examination of the stools yields valuable evidence if undertaken early in the disease, but if delayed until after the second week, when necrotic changes have taken place in the ulcers, the predominance of putrefactive organisms renders the results less reliable. For the purpose a drachm or so of the evacuation should be sealed in a test-tube and dispatched to the laboratory. The search for bacilli in the urine, on the other hand, is best undertaken at a later stage of the disease, when bacilluria is more common. The urine should be collected with aseptic precautions, being passed into a previously sterilized vessel and sent off at once to the bacteriologist.

The examination of the *rose spots* for bacilli, though of little practical value in the diagnosis of typhoid, since the presence of spots alone is sufficiently diagnostic, may be useful in the differentiation of typhoid from paratyphoid fever. The examination is effected by making a crucial incision in the spot, after freezing it, and scooping out the interior with a corneal spud or curette, the contents being then transferred to broth and incubated.

The *Widal reaction* (see SEROLOGICAL DIAGNOSIS) is a diagnostic method much in favour at the present day. Though less accurate than the identification of the bacillus in the blood or excretions, it is reliable in the large majority of cases, and is far more convenient to the busy practitioner who is called upon to obtain the material for examination. Unfortunately, the serum reaction is not obtainable in most cases of typhoid fever until the middle of the second week, though sometimes appearing a few days earlier, but by the fourteenth day it may be confidently expected. Should no positive result be obtained by the middle or latter end of the third week, the case is almost certainly not typhoid, though in very grave attacks, it is true, the reaction may be absent throughout. So reliable is the test that in not more than 2 per cent. (Ker) of cases of undoubted typhoid does a positive reaction fail to appear.

## TYPHOID AND PARATYPHOID FEVERS

at some period of the illness. The reaction usually remains positive for several months after the attack, occasionally for several years, and preventive inoculation is just as effective in causing agglutination as an attack of the disease itself. The extension of preventive inoculation with the triple vaccine (T A B) has greatly reduced the value of the agglutination reaction in the diagnosis of typhoid and paratyphoid fevers, though Dreyer's modification of the serum test is certainly of value. This consists in observing the sharpness in the rise and fall of the agglutination titre curve, which in the case of the homologous organism is usually at its maximum on the sixteenth day, in a consecutive series of examinations.

**Leucocyte count.**—Having regard to the frequent occurrence of leucopenia in enteric fever, a leucocyte count may yield some assistance in the diagnosis. Although there are exceptions to the rule, a fall to 5,000 or less is highly suggestive. In these circumstances a relative excess of the mononuclear cells is usually to be noted after the first week.

**Diazo reaction of Ehrlich.** This urinary test is of considerable value during the earlier stage of typhoid before the serum reaction is available (see URINE, EXAMINATION OF, p. 452). As Ker points out, the chief value of the test lies in its negative aspect, since both in tuberculosis and pneumonia, diseases which are liable to be mistaken for enteric fever at an early stage, the reaction is almost constantly positive; but it very rarely fails to be obtained between the sixth and twelfth day in a case of typhoid fever.

**Methylene-blue test of Russo.**—This test also depends on a colour change in the urine. Five drops of a 1-in-1,000 solution of methylene-blue are added to the urine, previously filtered if turbid. An emerald-green tint is imparted to the urine if the result is positive. Though often disappointing, the test is regarded by one of us (J. D. R.) as a more reliable indication than the diazo reaction, and as being of some value in prognosis.

The diseases most likely to be confused with enteric fever may be said to fall into one of two groups, the resemblance in the one case attaching to the presence of continued fever, unattended at an early stage with any ascertainable inflammatory lesion of an organ or other structure, and in the other case to the presence of obvious intra-abdominal inflammation.

The first group includes certain general

febrile diseases, for the most part of an infective character, i.e. typhus, influenza, pneumonia, broncho-pneumonia (especially in children, in whom physical signs in the chest may be inconspicuous throughout), acute miliary tuberculosis, tuberculous meningitis, cerebrospinal fever, infective endocarditis, and sepsis.

The second group comprises tuberculous peritonitis, enteritis, gastro-intestinal catarrh, colitis, dysentery, appendicitis, and pelvic suppuration in women.

For the purpose of differentiation, attention to the foregoing particulars should be of assistance.

**Mortality and prognosis.**—The mortality differs considerably in the different age-periods. It is lowest among patients between 5 and 10 years of age, and then increases with each quinquennium until it amounts to 40 per cent. at the age of 60 and onwards. The mortality is somewhat greater in females than in males.

Among inoculated patients the mortality has fallen to 1 or 0.5 per cent. (Goldscheider).

In forming a prognosis, the previous health of the patient must be considered. Specially unfavourable are alcoholism, chronic pulmonary or renal disease, obesity, and pregnancy.

**Temperature.**—The height and duration of the continued fever, though of less importance than the pulse, nevertheless afford valuable prognostic guidance. A morning temperature of 104° F. throughout the second week indicates a very severe attack. The same may be said of a temperature which, after the second week, shows no marked morning remission or is little affected by cold baths, tepid sponging, or other antipyretic treatment. As a rule, the greater the remissions the milder the case. A sudden fall of temperature suggests collapse which may be due to intestinal hæmorrhage. The occurrence of rigors during the height of the fever is not very uncommon. Though the temperature may reach 105° F., and the condition appear threatening, their import is not unfavourable as a rule.

**Cardio-vascular system.**—A strong and slow pulse of 80–90 is a favourable sign, while a pulse-rate persistently over 120 in an adult is of bad omen. A rapid pulse in a woman or child has less significance. The blood-pressure is of less importance prognostically than the frequency of the pulse. Typhoid fever tends to depress the arterial tension more than any other acute infectious disease, and low records are often met with in quite mild cases. A



## TYPHOID AND PARATYPHOID FEVERS

*sudden* change, however, in the blood-pressure in a downward or upward direction is important. The former indicates collapse, and the latter suggests a complication, especially peritonitis, in which readings varying from 156 to 208 mm. Hg have been recorded, as compared with a mean pressure of 104 among all typhoid cases (Crile). On auscultation of the heart, weakness or disappearance of the first sound, embryocardia, and "gallop rhythm" are of evil omen.

**Alimentary system.**—In favourable cases the tongue keeps moist and is only lightly furred. A dry, coated, and swollen tongue little affected by constant attention is a grave sign. Meteorism is also of unfavourable significance. Not only does it indicate a loss of tone in the intestinal wall, but it tends to interfere with the cardiac and respiratory functions, and is likely to favour perforation.

The character of the stools is, as a rule, a good indication of the progress of the case, the liquid discharges becoming semiformed and finally solid as convalescence approaches. The presence of definite sloughs in the stools during the third and fourth week is always a source of anxiety, since it connotes a risk of hæmorrhage or perforation.

The condition of the abdominal reflex, which is affected in a large percentage of all cases of typhoid fever, may be considered in this connexion. The return of a lost reflex and, *a fortiori*, resumption of its normal activity, are favourable signs, indicating that the intestinal lesions are undergoing repair; they usually accompany the change in the character of the stools. Persistent diarrhœa is of bad omen.

Intestinal hæmorrhage, if profuse, is a grave symptom, not only on account of the anæmia it entails, but also because it indicates deep ulceration and often heralds the onset of perforation. According to Osler, 35 to 50 per cent. of the cases of intestinal hæmorrhage are ultimately fatal.

Perforation is almost unavoidably fatal unless early and skilful surgical treatment be obtained.

**Nervous system.**—The importance of nervous symptoms must be determined by the ordinary nervous stability of the patient. Symptoms which would justifiably cause alarm in the case of a person with normal nervous equilibrium are of less importance in a neuropathic subject. Quiet delirium occurring only at night has no untoward significance; delirium during

the morning is more serious. Subsultus tendinum, carphology, incontinence of urine and fæces, and trophic disturbance of the skin shown by rapid development of bedsores, indicate a severe attack.

The prognosis of meningeal symptoms can only be settled by lumbar puncture. The outlook in meningism or serous meningitis is not necessarily unfavourable, while in purulent meningitis it is exceedingly grave.

**Diazo reaction.**—Some prognostic significance may be attached to the behaviour of the diazo reaction. In all but severe attacks it tends to become negative in the course of the second and third weeks. A persistence of a positive reaction beyond this period indicates a severe attack. A sudden disappearance of the reaction associated with deterioration of the general condition is also of bad omen.

**Treatment. Prophylactic.**—The chief safeguards against an attack of typhoid fever consist in the maintenance of a pure water supply, the provision of clean milk, the control of the shellfish and ice-cream trades, the proper disposal of sewage, and the destruction of flies and their larvæ.

On the occurrence of one or more cases the patients must be isolated, their excreta disinfected, and a search for possible carriers instituted.

During an epidemic the milk and drinking-water should be boiled and the food protected from flies. All attending a typhoid patient must keep their hands and finger-nails scrupulously clean, and should never take their meals in the ward or sick-room.

**Inoculation.**—The above precautions, if adequately carried out, afford sufficient protection against typhoid fever, but it is not always possible to ensure them. Whenever there is any doubt as to the proper supervision of the sources of infection, inoculation should be employed. In the case of soldiers, especially, it should be made compulsory, as it has been in the United States Army since 1911, and in the French Army since 1914.

The principles of typhoid immunization and the methods of preparation of the vaccine are discussed under IMMUNITY. The vaccine should not be a simple typhoid vaccine but a mixed one, consisting, as Castellani recommends, of the typhoid bacillus, the paratyphoid-A bacillus and the paratyphoid-B bacillus. The reaction caused by this mixed vaccine is not more severe than that following the use of the simple typhoid vaccine, and the inoculated

## TYPHOID AND PARATYPHOID FEVERS

persons benefit by being protected against three diseases instead of only one.

In the great majority of cases inoculation confers complete immunity of from two to four years' duration. In the exceptional cases which develop fever of a typhoid type within this period, bacteriological examination of the blood, bile or fæces may show a paratyphoid organism, but usually the true typhoid bacillus is present (Carnot and Weil-Hallé). This lack of immunity has been attributed to various causes—to insufficient doses, to too great an interval between the injections, to the inoculation being performed during the incubation period, to special circumstances connected with the individual, such as overwork, acute or chronic disease, especially tuberculosis, or to an overwhelming number and virulence of the infecting organisms. But even when the immunity is not complete, the attack of typhoid fever is usually mild and uncomplicated.

Inoculation is contraindicated in the presence of acute or chronic disease, especially tuberculosis, diabetes, and the acute stage of syphilis, and is less urgent in the case of persons above 40, owing to the comparative rarity of the disease at that age, than in younger persons.

**Curative.** *General considerations.*—In no disease is it more important, but unfortunately less frequent, for the patient to come under treatment at an early stage. Every person suspected to be suffering from typhoid fever should at once be put to bed and, if possible, isolated. Unless adequate nursing and isolation can be secured at home, he should be removed to hospital without delay.

If he is to be kept at home, two good nurses must be obtained for day and night duty respectively; the sick-room should be large, quiet, and well-ventilated, but free from draughts to prevent aggravation of the bronchitis which is such a constant feature of the disease, and an even temperature of about 60° F. should be maintained. The bed should be narrow and accessible from each side. It is often advisable to have two beds for day and night use respectively. Except in cold weather a single sheet and one blanket at most are sufficient covering. The night-shirt should open down the back and be fastened with tapes. A vest is not required.

The temperature should be taken four-hourly until the evening record is normal, and then every morning and evening for the follow-

ing month. If the temperature rises again, a four-hourly chart should be resumed until the evening temperature has become normal again. The pulse and respiration should be charted simultaneously with the temperature.

The patient should not be allowed to sit up for any purpose until at least a week after the evening temperature has become normal, and independent movements, such as turning from side to side, should be discouraged during the febrile period. Every change of position should be left to the nurse. Too long a stay in one position, especially in the dorsal decubitus, not only favours the development of bedsores, but is apt, especially in severe cases, to give rise to hypostatic pneumonia. The pressure exercised by a prolonged lateral decubitus has been held responsible for some cases of typhoid ulnar neuritis.

The skin should be thoroughly washed with soap and water every morning and evening. The back should be rubbed with methylated spirit and dusted with starch. Reddened and abraded areas should be treated with zinc ointment or lanolin, and a water bed should be provided.

Special care should be devoted to the mouth, teeth, gums, and palate, which should be cleansed with a tooth-brush or a piece of lint wrapped round the finger and dipped in a mouth-wash such as the following:—

R̄ Pot. chlor. gr. x.  
Tr. lavand. ℥x.  
Glycer. bor. ʒi.  
Aq. ad ʒi.

The bowels should be moved every second or at least every third day. It is advisable, especially after the first week, to abstain from the oral administration of any laxative and to open the bowels when necessary by a soap-and-water enema. When the temperature has been normal for about a week, a dose of castor oil may be substituted.

Retention of urine, which is not infrequently met with in male adults in the early stage of the disease after the recumbent posture has been enforced, may be obviated by the application of hot fomentations to the hypogastrium; but it may be necessary to pass a catheter morning and evening for three or four days before the retention disappears. The administration of urotropine, 5–10 gr. four-hourly, is advisable to prevent urinary sepsis, but its tendency to cause hæmaturia should be borne

## TYPHOID AND PARATYPHOID FEVERS

in mind and the drug discontinued on the first appearance of this symptom.

*Vaccine treatment.*—The vaccine treatment of typhoid fever, though less uniformly successful than preventive inoculation, is sufficiently efficacious to deserve special consideration. The best results are obtained if the treatment is begun before the tenth day. The preparations and doses vary considerably with different authorities. Krumbhaar and Richardson recommend 500 millions as the initial dose for an average adult, followed by two or more larger doses at three-day intervals. Leishman recommends smaller doses—100 to 250 millions. For children the doses range from 50 to 100 millions. As a rule, four or five doses are sufficient. The injections are usually given subcutaneously, but more rapid results are claimed from intravenous injection (Ichikawa).

The injection is usually followed by the appearance of a painful erythematous nodule at the site of puncture, enlargement of the adjacent lymphatic glands and of the spleen, rise of temperature and leucocytosis, and occasionally by diarrhoea and albuminuria; but these phenomena are all transitory.

The general condition is usually considerably improved; the temperature rapidly changes from continuous to intermittent, the duration of the fever is shortened, and perforation and relapses are rendered less frequent.

The mortality is comparatively low in cases treated by vaccines. In 2,256 such cases the death-rate was 5.71 per cent. (Gauchery).

About one-quarter of the cases are not affected by vaccines, either as regards the course of the disease or the attenuation of symptoms.

Their use is contraindicated in hæmorrhage or suspected perforation, or in such complications as pneumonia or otitis, where other organisms are involved (Krumbhaar and Richardson).

*Diet.*—There has been a general tendency in recent years to adopt a more liberal diet than was formerly permitted. The substitution of such a diet has been found to be well borne, to be attended by a low mortality, and not to entail a larger percentage of cases of intestinal hæmorrhage or perforation. The general condition throughout the disease compares very favourably with that of the patient on the ordinary milk diet, the typhoid state during the febrile period and exhaustive psychoses in convalescence being but rarely seen. Coleman

even claims that his high-calorie diet often renders bath treatment unnecessary.

The fluid diet to which the patient used to be restricted is founded on several false assumptions. It was supposed that the typhoid patient was unable to digest solid food and that its ingestion tended to raise the temperature and rendered intestinal hæmorrhage and perforation more likely to occur. It is important to realize that solid food, if digestible, is in liquid form by the time it reaches the ileum, and therefore can have no injurious effect upon the ulcers in that region.

Our aim should be to find a diet which will prevent loss of tissue without overtaxing the digestive powers of the patient. Protein may be supplied in the form of milk and eggs, but it is better to prevent the loss of pre-existing protein by increasing the amount of carbohydrate and fat than by adding fresh protein to the diet. The administration of new protein in the form of meat during the acute stage should particularly be avoided, for the putrefactive changes that such food undergoes in the intestine are liable to give rise to digestive disturbances, and the excretion of the toxic bodies thus formed may irritate the kidneys and produce nephritis. Meat extracts should be used carefully for the same reasons, but may be given for the sake of variety and to stimulate the appetite. Carbohydrates may be given in the form of lactose, cereals, bread or toast, breakfast rusks, boiled rice, or baked or mashed potatoes. Fats are not well tolerated by some patients, in whom they cause eructation, nausea, vomiting, and diarrhoea, especially in the early stages of the disease. In suitable cases fat can be given as cream, butter, and yolk of eggs.

No hard-and-fast rule can be laid down. Each patient must be treated as an individual, due regard being shown to his likes and dislikes for particular articles of food.

A good practical rule is to place each patient, when he first comes under treatment, on a milk diet for at least twenty-four hours, at the end of which time additions and modifications may be made. The amount of milk in the twenty-four hours should be about  $2\frac{1}{2}$  pints, which should be given diluted with plain water or barley water every two hours by day and every four hours by night. If curds be found in the stools, an equal portion of lime water should be added, or the milk should be peptonized or citrated by the addition of sodium citrate, 2 gr. to each ounce of milk. Milk should

## TYPHOID AND PARATYPHOID FEVERS

never be given to quench thirst, but should always be regarded as a food. On the other hand, the patient should be encouraged to drink as much cold water as possible, either in the form of pure water or as lemonade, to help in the elimination of the toxins. A-rated waters should be forbidden owing to their tendency to cause distension.

The best guide to increase in the diet is the appearance of the tongue and the condition of the appetite. If the tongue keeps moist and the appetite good, such articles as bread and butter, toast and butter, eggs, apple sauce and mashed potatoes may be allowed from the first.

The gradual return to normal in the character of the stools, and the resumption of its normal activity on the part of a previously absent or sluggish abdominal reflex, are indications that a further increase in the diet may be made.

Although there may be difficulty in determining whether it is due to an article of diet or to the toxæmia of the disease, the occurrence of digestive disturbance, such as increase in the diarrhoea and, still more so, abdominal pain or meteorism, should be followed by curtailment of any food substance likely to be responsible, or by its entire omission from the diet.

Intestinal hæmorrhage of considerable degree, and *a fortiori* perforation, call for immediate and complete suppression of all food.

In the early stage of the disease, and before any remission of the fever has taken place, it may be impossible to give enough food to prevent considerable tissue waste. At a later stage, when the appetite returns, the amount of the food can be increased and the loss of weight checked.

Coleman states that in most of the severe cases of typhoid it is not possible to give more than 3,000 Calories until the temperature passes into the steep-curve period, when the number of Calories may be increased to 4,000 or 6,000.

**Antipyretic treatment.**—A mild or moderate degree of pyrexia should not be interfered with, but should be regarded as the natural reaction of the organism to bacterial invasion. Antipyretic drugs, especially members of the coal-tar series, should be avoided, or used only occasionally and with caution, owing to their depressing effect. Quinine is safer, but has no specific action. It does not shorten the duration of the disease, and is liable to set up vomiting.

The control of pyrexia is much better carried out by the various methods of hydrotherapy, viz.:

1. **Cold baths.**—The patient is immersed in a bath at a temperature of about 65° F. and kept there for from fifteen to twenty minutes, during which time cold water is poured over his neck and shoulders, and he is told to rub himself and is rubbed by the attendant. A less rigorous plan is to put the patient in a bath of an initial temperature of 95° F., which should be gradually lowered by the addition of cold water and ice to about 70°. The benefits claimed from the cold bath are the reduction of temperature, the sedative effect on the nervous system, stimulation of the respiratory movements, rise of blood-pressure, and increased excretion of toxins through the skin and kidney. The objections to the cold bath are that it is cumbersome, is often strongly objected to by the patient and his friends, and not infrequently is followed by a relapse. It is contraindicated during collapse, intestinal hæmorrhage or perforation, and is unnecessary in mild or moderate cases.

2. **Cold pack.**—This is a less drastic method than the cold bath, and is in our opinion the best means of reducing hyperpyrexia. The whole surface of the body should be enveloped in a couple of drawsheets wrung out of cold or ice-cold water. After a period ranging from fifteen to thirty minutes, according to the strength of the pulse and the degree of cyanosis, the pack should be removed and the patient wrapped in a warm, dry blanket and, as soon as he is warm, laid between the sheets and covered with a single blanket.

3. **Trpid sponging.**—This is chiefly valuable as a nervous sedative, and is especially useful in cases of restlessness and insomnia. As an antipyretic it is inferior to the cold bath or cold pack. For the success of the method, it is essential that the sponge should be charged as full as it will hold, and the whole surface of the body, and especially the back, should be soused with the fluid.

The frequent administration of small quantities of cold water by mouth and the rectal injection of ice-cold water are other hydrotherapeutic methods which may be of value.

**Antiseptic treatment.** Antiseptics have no power to shorten the disease or to prevent such complications as hæmorrhage or perforation, or relapses. On the other hand, even if it be impossible to destroy the typhoid bacillus

## TYPHOID AND PARATYPHOID FEVERS

in the intestine except by poisonous doses, the propagation both of the typhoid bacillus and of the various putrefactive organisms may be somewhat held in check by the administration of certain drugs. Among these we have found oil of cinnamon to be the most useful for controlling abdominal pain, meteorism, and fœtor of the stools. It acts not only as an intestinal disinfectant, but also as a sedative to the nervous system. It may be given in 2½- to 5-min. doses every two to three hours throughout the disease, either as an emulsion or, if it tends to provoke vomiting, in gelatin capsules. Another useful drug is sulphurous acid, which may be prescribed as follows:—

℞ Acid. sulphuros. ℥xx.  
Syr. limonis ℥x.  
Aq. chlorof. ad ʒi.  
ʒi. quartis horis.

Oil of turpentine, in 5- to 10-min. doses in capsules or perles, is warmly recommended by some authorities, but should be used with caution owing to its irritating action on the kidneys and bladder.

**Treatment of special symptoms.** *Diarrhœa*.—When the motions exceed more than four in the twenty-four hours the diet will require some modification. Beef tea or meat juice should be stopped and the milk should be more diluted or peptonized. If the diarrhœa continues unaffected, the administration of salicylate or subnitrate of bismuth in 15- or 20-gr. doses every four hours is likely to be effective.

*Constipation*.—Purgatives should be avoided after the end of the first week and a soap-and-water enema every second or third day substituted. If the constipation is troublesome the addition of beef tea or meat juice to the diet is advisable.

*Meteorism*.—The abdomen should be examined daily for the presence of this symptom, which is not only a source of great discomfort and liable to give rise to perforation, but also interferes with the action of the heart and respiration. If possible, the part of the alimentary canal affected should be determined. Distension of the stomach when the patient cannot relieve himself by evacuation can be remedied by the passage of a stomach-tube. When the colon is affected, the passage of a rectal tube high up the gut may be tried, though it seldom proves effectual.

Turpentine is often of service, and may be employed in the forms of stupes to the abdomen,

or internally in doses of 6–10 min. every three or four hours, or in rectal injections of 1 or 2 dr. twice daily.

The value of cinnamon oil as a prophylactic is shown by the fact that, with the exception of patients in whom the condition was present on admission to hospital, no single instance of meteorism occurred among over 600 cases which one of us (F. F. C.) has treated with this drug.

Puncture of the distended gut is a dangerous proceeding, and is not to be recommended.

*Hæmorrhage*.—A slight amount of intestinal hæmorrhage, especially in a young and vigorous subject and occurring early in the disease, calls for no treatment. When the bleeding exceeds five ounces, and still more so when it is repeated, absolute rest should be enforced. The patient should be kept in one position, preferably the dorsal decubitus, and should not be moved for any purpose. He should not be given a bed-pan, but should be induced to pass his motions into absorbent wool or carbolized tow. An ice-bag should be applied to the abdomen and an injection of ½–1 gr. of morphia given to ensure complete mental and bodily rest. A starch and opium enema (tr. opii 30 min., mucilag. amyli to 4 oz.) may be used as an alternative. Feeds should be discontinued altogether for some hours or until the following day, or at least be considerably curtailed and given cold. The patient may be allowed ice to suck, but no drink of any kind should be given.

If there is collapse, the foot of the bed should be raised, and normal saline solution injected hypodermically.

As a hæmostatic, gelatin may be given subcutaneously or in an enema. Calcium lactate, in 10-gr. doses t.d.s., may be of value.

*Perforation*.—As soon as the diagnosis is made, surgical interference is imperative. According to G. E. Armstrong of Montreal, who closed 22 typhoid perforations with 11 recoveries, the following symptoms call for immediate abdominal incision, viz. persistent pain, definite change for the worse in the patient's expression, abdominal or rectal tenderness, rounding up of the abdomen, and increased resistance to pressure. If these symptoms are present, even if the temperature and pulse are not decidedly altered nor vomiting present, Armstrong regards the likelihood of perforation as very great.

*Headache*.—The headache, which is apt to be especially severe during the first week of the

## TYPHOID AND PARATYPHOID FEVERS

disease, may be treated by cold compresses to which have been added a few drops of vinegar, alcohol, or eau-de-cologne. Small doses of phenacetin, ammonol, or pyramidon may be given. The application of one or two leeches to the temples often affords immediate relief. Lumbar puncture has been recommended for cases in which the headache is unusually severe and persistent.

**Insomnia.**—If this symptom does not yield to hydrotherapy, various drugs may be administered, such as trional 15–20 gr., chloral-amide 30–40 gr., Dover's powder 5–10 gr., or a combination of sodium bromide, 15 gr., and chloral-hydrate, 8 gr. Alcohol is sometimes useful, especially when insomnia is due to exhaustion.

Insomnia and restlessness are sometimes due to constipation, in which case relief may be secured by means of a simple enema.

**Delirium.**—Mention has already been made of the sedative effect of the cold bath and of the regular administration of cinnamon oil internally. In severe cases constant observation on the part of the nurse is necessary to prevent the patient getting out of bed, and it will be often necessary to fasten him in with a draw-sheet. A hypodermic injection of morphia may be required to save the brain from exhaustion and to a certain extent to obviate the risk of hæmorrhage and perforation.

Typhoid delirium, especially in children, is often associated with meningeal irritation. In such cases considerable benefit may be derived from lumbar puncture, which relieves the pressure on the nerve-centres and evacuates a certain number of the bacilli and their toxins.

**Cardio-vascular symptoms.**—Careful examination of the heart and pulse is of the utmost importance in the treatment of typhoid fever, as in at least one-half of the fatal cases death is directly due to circulatory failure. A pulse exceeding 120 in an adult, especially when accompanied by weakness of the first sound, is a danger-signal calling for the cautious administration of stimulants. A feeble pulse is sometimes associated with marked ventricular dilatation, which is liable to be followed by pulmonary congestion, and in such cases alcohol is usually of value.

The routine use of alcohol in typhoid fever should be avoided, and it is advisable to keep it for the following emergencies: constant delirium and sleeplessness—with muscular

tremor, feeble circulation and a dry, brown tongue, undue weakness of the pulse without any other sign, cardiac dilatation, cyanosis, pulmonary congestion, and pneumonia.

### PARATYPHOID FEVER.

Though due to infection by organisms distinct from the typhoid bacillus and known as *B. paratyphosus*-A and -B, paratyphoid fever is indistinguishable in its clinical appearances from genuine typhoid fever. For all practical purposes, therefore, whether of prevention or treatment, it may be regarded as a variety of enteric fever. The disease is usually milder in its manifestations and tends to be less protracted, and although ulceration of the lymphoid glands in the bowel would appear to be the rule, it is probable that in many cases the inflammation of the affected follicles is limited to swelling merely, and stops short of actual ulceration. This, though difficult of proof, may be inferred from the slighter and less protracted fever, absence of sloughs from the stools, and the comparative infrequency of either hæmorrhage or perforation.

The following, amongst other points, have recently been claimed by some observers as characteristic of paratyphoid, viz. a more sudden onset, often with vomiting and shivering, headache, localized especially in the occiput, facial herpes, sweating, and phlebitis. These data, however, seem to be very unreliable, reports from different observers being most conflicting. That the pyrexia tends to run a more irregular course and be less prolonged in paratyphoid is, however, generally conceded. This seems to be especially true of paratyphoid A, in which a sudden, though temporary rise to a high degree is not uncommon in the early stage of the fever.

The lesions found post mortem may be identical with those in typhoid. The intestinal lesions, however, are apt to be very variable; in some cases they resemble those of typhoid; in others, those of dysentery, acute enteritis, or septicæmia, or there may be no changes at all in the intestine. The large bowel is more frequently affected, especially in paratyphoid B, than in typhoid. On the other hand, ulceration of Peyer's patches and perforation peritonitis are less frequent in either form of paratyphoid.

F. FOORD CAIGER.  
J. D. ROLLESTON.

## TYPHUS FEVER

**TYPHUS FEVER.**—An acute specific infectious disease, characterized by sudden onset, a continuous fever of about a fortnight's duration, a mulberry rash, and rapid defervescence.

**Etiology.**—The most active predisposing causes are overcrowding, physical and mental exhaustion, poverty and famine, which all tend to reduce the organic resistance and render the system more susceptible to contagion. The disease is therefore most frequent in war time, especially among prisoners and refugees and in invaded territories, as was exemplified in Serbia, Rumania, and Poland.

Typhus is chiefly confined to cold and temperate climates. At the present time it is most prevalent in Poland, Russia, the cooler parts of Asia, Northern Africa, and Mexico.

The disease is more common during the cold season, but, as Murchison pointed out long ago, its greater incidence at this time is not referable to mere cold but to the protracted overcrowding and more defective ventilation, to which may be added the greater possibilities for dissemination by infected lice. It is most frequent and characteristic in adults, but children, in whom it usually assumes a mild form, are by no means exempt, and it is probable that a more general employment of the Weil-Felix reaction (*see* Diagnosis) will prove that the disease is much more common in childhood than has hitherto been supposed. The incidence is approximately the same in the two sexes.

Typhus is a lice-borne disease. It is transmitted by the louse, and the louse only. A single bite is sufficient for infection. The body-louse is by far the most usual agent of transmission, but head-lice can also convey the disease. The crab-louse has no etiological significance. The louse becomes contaminated during the febrile period after the fourth day, but chiefly between the fifth and seventh day. The blood of convalescents is not infectious. An infected louse, however, retains the virus in a virulent form at least up to the twenty-fourth day, and probably throughout its life. Healthy immune persons who are carriers of lice can thus play as important a part in the dissemination of the disease as the actual typhus patient.

Though complete agreement does not yet exist with regard to the microbiology of the disease, the causal agent is probably a dumb-bell-shaped protozoon known as the *Rickettsia prowazeki*, so called, after the investigators who

lost their lives in the study of the disease, by Da Rocha Lima, who found it in large quantities in the gastro-intestinal canal of infected lice. Cultures both on aerobic and anaerobic media are negative, and the *Rickettsia* can only be kept alive by feeding the lice either on convalescents or with the blood of a sucking-pig. The germ is not transmitted from man to man, but from man to louse and from louse to man. The louse is an intermediate host in which the organism undergoes a transformation probably of the same kind as the malarial parasite in the *Anopheles*. According to Jaffé, the *Rickettsia* is introduced by the bite of the louse into the blood; there it circulates until it is taken up by the endothelial cells of the capillaries or smallest arterioles, in which it multiplies and causes an irritation of the surrounding tissue that reacts by the formation of a typhus nodule.

**Pathology.**—There are no characteristic naked-eye changes in typhus post mortem, such lesions as are present being those usually found in the other acute exanthemata. In the early stages the spleen is always enlarged and extremely soft; later it becomes firmer. The kidneys show punctiform hæmorrhages. The heart is dilated and presents all the signs of infective myocarditis. Endocarditis and pericarditis are very rare. The diaphragm and stomach are usually free from characteristic changes. The mucous membrane of the intestine shows catarrh and frequently ecchymoses, but Peyer's patches are not infiltrated and intestinal ulcers do not occur. Bronchopneumonia is the most frequent lesion found in the lungs. The liver shows no characteristic changes, and the brain is normal on naked-eye examination. On the other hand, the microscopical lesions are pathognomonic. Recent investigations have shown that typhus is anatomically a systemic disease of the small vessels. The characteristic lesion is the typhus nodule, which is formed by necrosis and proliferation of the endothelium associated with perivascular infiltration of leucocytes and other cells. The lesions are most pronounced in the skin and central nervous system, and are present in a less degree in the myocardium and practically all the other organs. Most of the nodules disappear entirely, but in the brain a few become replaced by small cicatrices.

**Blood.**—According to Rothacker, the blood picture of typhus is not typical, and varies in the different stages of the disease. During the first four days it is usually normal; and in

## TYPHUS FEVER

the eruptive stage the relative percentage of polymorphonuclears is increased, though the total number of leucocytes remains normal. From the tenth day onwards the blood picture entirely changes. The total number of leucocytes is usually between 10,000 and 14,000 per c.mm., and is entirely independent of the severity of the disease. The polymorphonuclears are 95-97 per cent., and lymphocytes are as low as 3-6 per cent. Eosinophils are absent. The red cells often number only 2-3,000,000 per c.mm. in convalescence. The leucocytes in many cases show a considerable increase. Eosinophils reappear, and lymphocytosis occurs. By the eighth to the tenth day of convalescence the blood picture is normal again.

**Symptomatology. General.**--The incubation period is usually about twelve days, but may vary from a few hours to over three weeks. In cases of medical men who, after being bitten by a louse, have noted carefully the date of onset, the incubation period has ranged from seven to twenty-three days. Rises of temperature during the incubation period are not uncommon, as has been found by systematic observation of contacts.

The onset is usually quite sudden, with a rigor or feeling of chilliness. The temperature rises very rapidly and reaches 102.4-104° F. within twenty-four hours. The patient feels extremely languid and, if possible, takes to bed at once, suffering from severe headache and vertigo. The face is turgid. Conjunctival injection is an early and characteristic feature, and hæmorrhages in the conjunctiva appear before those in the skin. The tongue is thickly coated, with the tip and edges free; later it becomes dry and brown, forming the "parrot tongue" of typhus. Nausea is sometimes complained of, but vomiting is rare. The bowels are usually constipated, and diarrhoea is exceptional. The pulse is 100-120, the respirations are 30-40, and there is a frequent distressing cough due to the presence of bronchitis. The skin exhales a peculiar odour compared to the smell of mice or rotten straw, but best described as *sui generis*.

The urine, as a rule, contains slight traces of albumin, and the diazo reaction is almost always positive. The chlorides are greatly reduced or entirely absent. The temperature reaches its maximum by about the fourth day, and remains high until the fourteenth day, when in uncomplicated cases it falls rapidly, reaching normal within one or two days. Before the temperature becomes finally settled

there is often a precritical rise to 104° F. or above, and a pseudo-crisis is almost as frequent. The intermissions which so frequently anticipate defervescence in typhoid fever do not occur. Death is often preceded by hyperpyrexia.

The eruption appears between the fourth and seventh days and lasts from seven to ten days; it is usually first seen on the sides of the chest and flanks, and then extends all over the trunk, invading the limbs last of all. Occasionally it appears upon the face. It first consists of very slightly raised dusky pink spots of irregular outline, closely resembling measles, but in from one to three days the spots grow darker and a considerable proportion become definitely petechial. Other spots are associated with a less distinct subcuticular mottling, the association of the two elements being known as the "mulberry rash" of typhus. The characteristic eruption is sometimes preceded by an initial erythema on the face and forearms on the third or fourth day. During the second week the nervous manifestations become more or less marked. As a rule, symptoms of nervous excitement are most pronounced towards evening and in the night time, and the prostration is greatest in the morning (Murchison). Subsequently the prostration becomes more severe, and the patient remains in a stuporous state until about the fourteenth day, when in favourable cases the fall of temperature is accompanied by a remarkable improvement in the general condition. Convalescence is usually slow.

Relapses and second attacks are very rare.

Although, as a rule, typhus is less subject to variation than other acute exanthemata, anomalous forms are occasionally met with. The conjunctivitis, nervous symptoms, and even the eruption may be absent, especially in children, and the fever in such cases is usually of short duration. On the other hand, the disease may run a fulminating course and prove fatal in two or three days. Prolongation of the fever to three or four weeks is usually due to a pulmonary complication.

**Respiratory system.** - Catarrh of the nose, naso-pharynx, larynx, trachea, and bronchi is almost constant at the height of the disease. The larynx may be affected by oedema, ulceration, and necrosis, which is apt to be followed by stenosis. Broncho-pneumonia often occurs, and hypostatic congestion is frequent in severe cases. Lobar pneumonia is rare.

**Cardio-vascular system.** --Myocarditis, which



## TYPHUS FEVER

is manifested by weak and distant heart-sounds and a feeble and irregular pulse, is practically the only cardiac complication of typhus. Endocarditis and pericarditis are very rare. Venous thrombosis and arterial thrombosis and embolism are frequent in some epidemics. The blood-pressure falls about the end of the first week, and continues to do so during the following week; in fatal cases the pressure drops until death. In convalescence it slowly rises, but takes a long time to reach normal (Danielopolu).

**Urogenital system.**—In rare cases acute parenchymatous nephritis develops and may give rise to fatal uræmia. Orchitis is rare. Gangrene of the external genitals is frequent in some epidemics. The menstrual periods, as in other infectious diseases, often occur prematurely at the onset or may be absent during the disease. Typhus has little, if any, influence on the course of pregnancy.

**Nervous system.**—The knee-jerks usually disappear about the fifth day of the disease, and remain lost throughout the febrile stage and for a varying period after this has subsided (Willcox). In convalescence they may be exaggerated, and ankle-clonus is sometimes found. As a rule, the abdominal and cremasteric reflexes are also lost during the febrile stage. Headache, which is often extremely violent, general hyperæsthesia, insomnia, restlessness, and delirium are constant symptoms in a typical case. Convulsions seldom occur. A few cases of hemiplegia have been recorded, and are probably due to the vascular changes described above. Paralysis due to lesions of the spinal cord or peripheral nerves are rarer than in enteric fever. Meningitis is seldom met with. During the acute stage the cerebro-spinal fluid is clear but under slightly increased tension, and shows an excess of albumin and leucocytes in which polymorphonuclears predominate. The Weil-Felix reaction is much less marked in the cerebro-spinal fluid than in the blood. The mental sequelæ are similar to those met with after enteric fever. Lack of concentration and inability for mental effort may persist for several months after an attack.

**Special senses.**—Deafness due to otitis media or to central changes is a fairly frequent symptom, but rapidly disappears in convalescence. During the febrile stage lesions of the iris, with secondary cataract, optic neuritis, and paralysis of the oculo-motor nerve or external rectus, may develop. More frequently ocular

complications are met with in convalescence, usually due to streptococcal infection, and consisting of palpebral abscess, inflammation of the orbit, corneal ulcer, and optic atrophy.

Other complications are inflammation and suppuration of the parotid, and abscesses in the joints and subcutaneous tissue.

**Diagnosis.**—During an epidemic the diagnosis of a typical case is easily made from the sudden onset, initial symptoms, and characteristic eruption. In sporadic cases considerable difficulty may arise. The initial symptoms resemble those of many other infectious diseases, and even the eruption is not always characteristic. In some cases in which the rash is discrete and punctiform the application of a cupping glass or an elastic band may render it more distinct. Very considerable value attaches to the Weil-Felix reaction, so called from the Austrian observers who first discovered it. The principle of the reaction is that the serum of a typhus case agglutinates the Bacillus X19, an organism of the *Proteus* group isolated from the patient's urine. The agglutination appears at about the fifth day of the disease, may occur in as high a dilution as 1 in 10,000 or more, and then diminishes, but persists after recovery for a period ranging from several weeks to several months. Normal serums or serums from other diseases than typhus do not agglutinate beyond 1 in 50, so that a well-marked agglutination at 1 in 50 or above is regarded as specific of typhus. The intensity of the agglutination bears no relation to the character of the typhus attack. The cerebro-spinal fluid possesses only a weak agglutinative power.

Children occasionally show no eruption, and the other symptoms, such as conjunctivitis, bronchitis, and nervous manifestations, may be absent. In such cases it is only by the temperature, a positive Weil-Felix reaction, and an association of the case with other cases in the family that a diagnosis can be made.

**Differential diagnosis.** *Enteric fever.*—The onset is usually much less sudden in this disease than in typhus. The conjunctivitis peculiar to typhus is absent, and the eruption appears later and is less profuse. In paratyphoid fever, however, the rash is apt to become generalized. The condition of the pulse, about 20 beats to the minute slower in enteric than in typhus, is a guide. Enlargement of the spleen and the character of the stools are also distinctive. In 80 per cent. or more of the cases typhoid bacilli can be found in the blood,

## TYPHUS FEVER

and at a later stage Widal's reaction and the presence of bacilli in the urine and faeces clear up the diagnosis. Lastly, in enteric there is usually a leucopenia, whereas in typhus leucocytosis is the rule.

**Smallpox.**—In both diseases there is a sudden onset accompanied by considerable prostration, and in the absence of the characteristic prodromal rashes of smallpox the diagnosis may be impossible during the initial stage. On the third day, however, in the case of smallpox the eruption appears and the temperature falls, whereas in typhus it remains high.

**Measles.**—The eruption is very similar to that of typhus, and, like it, appears on the fourth day, but is preceded by sneezing and other catarrhal signs and by Koplik's spots, which are absent in typhus.

**Influenza** may give rise to difficulty, especially if epidemics of both diseases are prevalent at the same time. The onset, however, is usually even more sudden in influenza than in typhus, and the temperature, apart from complications, rarely remains high for more than a few days.

**Apical or central pneumonia**, in which no local signs may be detected for several days in spite of careful examination, may also be mistaken for typhus.

**Malaria and relapsing fever**, which, in their initial stages, may resemble typhus, are differentiated by examination of the blood, which shows the presence of the malarial parasite in the one case, and of the spirochæte of relapsing fever in the other.

It must, however, be borne in mind that the association of diseases, though exceptional in peace time, is not rare in war. Thus typhus may be combined with enteric fever, dysentery, relapsing fever, or scurvy. In such cases the Weil-Felix reaction will be of great value.

**Prognosis.**—Age is perhaps the most important factor in deciding the issue of a case. The younger the patient the better the prognosis and the shorter the course of the disease. Typhus is rarely fatal until after puberty. The mortality up to 20 years of age is barely 5 per cent., while the danger to life increases rapidly with age, the mortality among persons over 50 being between 60-70 per cent. The mortality is higher among brain-workers, especially among medical men, than in the uncultured. Persons whose brains are damaged by alcohol run a special risk; but mental worry, and even fear of contagion, also have an unfavourable influence on the course of the dis-

ease. As regards the prognostic value of special symptoms, the condition of the nervous system is of greater importance than the temperature, though it is a bad sign when the temperature continues very high, or rises and falls during the second week. The early appearance of severe cerebral symptoms is always characteristic of a severe attack. Progressive increase of the pulse-rate is a grave sign, the crossing or convergence of the pulse and temperature being particularly unfavourable.

A leucocytosis of over 20,000 is of bad augury (Danielopolu). As a general rule, the severity and duration of the disease are directly related to the abundance and petechial character of the eruption. The development of complications is an unfavourable sign, especially broncho-pneumonia, parotitis, and bed-sores.

**Prophylaxis.**—Strict supervision of the passenger and goods traffic on railways and on ships coming from infected areas is imperative. A thorough destruction of lice on the skin and clothing of contacts must be carried out; their hair and beard should be cropped close, and the clothing disinfected or burnt. A system of notification and evacuation of infected quarters will be required. In addition to isolation hospitals for the sick, shelters must be established where contacts can be kept in quarantine for a period of at least fifteen days. Prophylactic inoculation with defibrinated blood taken from patients at the height of the disease or shortly after the temperature has become normal has been employed, but does not appear to confer any absolute protection against infection, though the course of the disease is milder, and the mortality less, in inoculated persons.

**Treatment.**—Generally speaking, the treatment of typhus is that of typhoid fever (q.v.). If possible, the patient should be nursed by attendants who have been rendered immune by an attack, and elderly nurses, among whom the mortality is high, should not be chosen. It is most important that the patient should have plenty of fresh air. Some authorities favour the nursing of the patient throughout the acute stage in the open air, which appears to have a soothing effect upon the nervous symptoms, especially the violent headache and insomnia of the initial stage.

Hydrotherapy is also beneficial in the form of cold baths, a cold-water mattress, or the local application of cold by an ice-cap or Leiter's tubes.

## ULCERATING GRANULOMA OF THE PUDENDA

The results of subcutaneous or intravenous injection of serum from convalescent patients have hitherto been disappointing, the mortality among cases so treated being about the same as among the controls. Treatment must be mainly symptomatic. The chief object is to maintain the patient's strength. Sir W. H. Willcox has emphasized the great value of administration of normal saline. In severe cases this should be given per rectum every four hours, beginning about the eighth day,

and followed in the later stages by subcutaneous and intravenous injections.

The diet should be the same as in all acute fevers. During the acute stage it should consist of fluids. If milk is not well borne, the same substitutes may be given as in enteric fever. Eggs, soup, or beef tea may be given in addition if the patient is willing and able to swallow. During convalescence a rapid increase in the diet is indicated.

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**ULCER, RODENT** (*see* SKIN, MALIGNANT GROWTHS OF).

**ULCERATING GRANULOMA OF THE PUDENDA.** (*syn.* Granuloma Venereum; Granuloma Inguinale).—An infective disease of tropical countries, whose sole manifestation is a peculiar granulomatous condition of the pudenda. It is met with chiefly in Brazil, India, Guiana, Papua, and northern Australia, sporadically in the southern United States, the West Coast of Africa, and Southern China.

**Etiology.**—The disease is almost certainly venereal in nature and conveyed by sexual contact, but the causal organism still awaits recognition, though several have been regarded as possibly culpable. An organism isolated by De Souza Araujo, and named by him *Calymmatobacterium granulomatis*, has since been studied by E. L. Walker, and is considered by him as probably the *Bacillus mucosus capsulatus*. There is considerable doubt whether it is responsible for more than a secondary infection. Commoner in women than in men, the disease is found only after puberty, and up to the age of 50.

**Symptomatology.**—A small raised nodular thickening appears on the genitalia or in the groin, frequently on the penis in the male and on the crura of the clitoris in the female. The skin or epithelial surface which covers it becomes attenuated, pinkish in colour, and easily excoriated or rubbed off, leaving a superficial ulcer, readily bleeding and extending, but attaining no considerable depth. Very slowly the disease progresses by continuous enlargement of the ulcerating area and by spreading

to opposing surfaces. Moist surfaces especially are invaded, so that it spreads along the groins, into the labia and vagina, and between the scrotum and thighs, but so slowly that a large ulcerating area is only developed after years. Healing and extensions take place concurrently, a dense, puckered cicatrix being formed in parts of the ulcer remote from the spreading edge. Despite its hardness this scar tissue may again be the site of active disease and ulcerate afresh. The appearance in a developed case is that of a voluminous, puckered and hard mass of unhealthy scar tissue, perhaps excoriated or breaking down, encircled by a narrow zone in which the characteristic granulomatous tissue can be seen as a nodular area, pinkish or red in colour, ulcerated and fissured. More remotely, discrete, raised nodules not yet denuded may be recognized. A peculiarly offensive watery discharge exudes from the ulcerated area, and may be profuse. Though the disease remains a local one, serious results attend its extension if it be allowed to continue for several years, for it ultimately invades the rectum and anus, the urethra, and the recto-vaginal septum. The general health, in spite of these eventualities, usually remains good. It is stated that the related lymphatic glands are only affected in women, in whom, too, sterility ensues.

**Diagnosis.**—From *malignant* and *syphilitic ulceration* in this region, granuloma venereum is easily distinguished by its extreme chronicity, its freedom from general symptoms, the absence of enlargement of lymphatic glands, and by failure to react to mercury and iodides. To *lupus vulgaris* it bears a closer resemblance, but is always confined to the pudenda, has none of

## ULCERATION

the histological features of tuberculosis, and tubercle bacilli are absent.

**Treatment.**—The older methods by scraping, caustics, the actual cautery, and free excision, the last of which proved to be most satisfactory, though often drastic, have been replaced by X-rays and intravenous injections of tartar emetic, either alone or in combination. Both these procedures have been very successful, except in occasional cases. At first 5 c.c. of a 1-per-cent. solution of tartar emetic in normal saline is advised, the dose being cautiously increased to 10 or 12 c.c. daily or every second day. A powder consisting of equal parts of antimony oxide, zinc oxide, and calomel may be dusted over the ulcer, or it may be dressed twice daily with compresses soaked in a 1-per-cent. solution of the tartar emetic. By these methods a cure is generally effected, though in the case of tartar emetic large doses may be necessary.

FREDERICK LANGMEAD.

**ULCERATION.**—An ulcer results from loss of substance at a superficies, and may be on the surface of the body or in a cavity lined by mucous membrane. Classified according to their causes, ulcers may be grouped thus:—

1. Traumatic.
2. Infective.
  - Pyogenic.
  - Chancroidal.
  - Syphilitic.
  - Tuberculous.
  - Actinomycotic.
  - Other specific ulcers, e.g. anthrax sores, yeld sores (q.v.), Bagdad boils (*see* ORIENTAL SORE).
3. Circulatory.
4. Neurogenic.
5. Thermal.
6. Malignant.

**Clinical features.**—The syphilitic ulcer with its characteristic circinate edge, steep sides, and base overlaid by a wash-leather slough; the tuberculous ulcer with its thin blue undermined edges, and base covered by flabby pale granulations; the actinomycotic ulcer with its very pronounced neighbouring induration and granule-containing discharge; the anthrax sore with its black central slough and usually severe general reaction; and the malignant ulcer, so obviously occurring in a new formation, so devoid of any attempt at repair, though there may be a few signs of a secondary infection—all these are fairly easily recognized, and are described under their

appropriate headings. The neurogenic ulcer, when it occurs as a perforating foot ulcer, as in *tabes dorsalis*, has also distinctive characters. There is the opening on the sole of the foot typically situated under the heads of the metatarsal bones, with little surrounding inflammatory reaction and with the epithelium around it heaped up into a white zone. These sores are anæsthetic, and a probe inserted will frequently impinge upon necrotic bone and a disorganized joint without the patient being aware of it. Moreover, signs of the antecedent nervous disease—*tabes*, syringomyelia, diabetic neuritis, general paralysis, etc.—will be found if searched for.

Most of the other ulcers resemble each other closely.

In the *spreading stage* ulcers have angry, inflamed, irregular edges; the base is shreddy with sloughs; there is no sign of granulation tissue. This appearance is altogether changed in the *healing stage*. Now the surface becomes covered with closely-packed small red protuberances (granulations), and the edge shows signs of centripetal epithelial spread—an outer white zone and an inner blue margin. These favourable features do not always persist until the whole sore is closed. They may change into those of a *stationary* ulcer, in which the surface is not covered by healing granulations, nor, indeed, by any proper granulations at all, but becomes smooth and red. The edges do not shelve down to the raw surface as in the healing ulcer, but are raised. The blue margin of advancing epithelium has gone. The discharge is scanty and watery, and the surrounding tissue, whilst it lacks the signs of acute inflammation, may be œdematous and pigmented.

There are several reasons why a healing ulcer becomes stationary. The general health of the patient may be poor; apart from acute general infections, the maladies which have the most baneful influence upon the healing of ulcers, and, indeed, of wounds or fractures of all kinds, are chronic interstitial nephritis and diabetes. Local impairment of circulation, such as the stasis dependent upon varicosities in the superficial veins, or that which occurs in the lower limbs of old persons or sufferers from weak cardiac action, has a harmful effect upon repair. Some large ulcers do not heal because of their size. Beyond a certain zone, repair stops because the vessels supplying the granulations become compressed by the contraction of the new-formed fibrous tissue through which they have to run. If the

## ULCERATION

base of an ulcer should become fixed to an underlying bone it cannot contract, as it should do during the healing process, and the ulcer becomes stationary.

Certain ulcers on the legs are extremely sensitive either over their whole surface or at definite points, and are called *irritable* ulcers. Ulcers overlying bone may, by their proximity, set up a local periostitis with new-bone formation. Epithelioma may grow from the edge of a chronic ulcer.

**Treatment.**—Generally speaking, ulcers should be treated by the methods described under WOUNDS, TREATMENT OF. In the infective stage the principles there laid down for combating infection must be followed. In the healing stage ulcers should be treated in the manner recommended for granulating wounds. Yet these methods are not always sufficient to induce stationary ulcers to heal; such intractable ulcers are usually leg ulcers, ulcers following extensive burns, X-ray burns, or trophic sores.

**Leg ulcers.**—The main adverse factor to be overcome is the impairment of the circulation, whether it be due to varicose veins or to feeble action of the heart. In either case the circulation through the legs will be improved if the patient lies up. Many *varicose* ulcers will heal if, in addition to ordinary wound treatment, the patient be kept in bed. If this measure is inconvenient or impossible the varicose veins must be supported in some other way. The best is the use of *Unna's paste*, which has the following formula:

R̄ Zinc oxid	. . .	3 parts.
Gelatini . . .	. . .	3 parts.
Glycerini . . .	. . .	5 parts.
Aquæ . . . . .	. . .	9 parts.

The ulcer is rendered as clean as possible, and a single layer of a gauze bandage is wound round the limb from the foot to the knee and is painted over with the melted paste. The procedure is continued until three, four, or five layers have been laid down. The paste sets and forms an elastic flexible support to the veins; it must be renewed every seven to ten days.

Again, the veins may be supported by adhesive strapping applied from the foot to the knee, a method which is more expensive and, the covering being impervious, less effective than *Unna's paste*. Elastic stockings are useful when new, but soon become inefficient through stretching. Martin's pure rubber bandage is convenient in that it can be washed

easily, but is undesirable because it is impervious to moisture. A *crêpe Velpeau* bandage is simple, elastic, and cheap. Next to *Unna's paste* it is the best means of supporting the distended veins. Always, in the ambulant treatment of varicose ulcers, the patient should be urged to seize every opportunity to aid the circulation through the lower extremities by posture. Whenever he is sitting he should rest his legs upon another chair. Standing should be avoided.

*Irritable* leg ulcers are a great source of pain and discomfort. A careful examination will show that there are one or more exquisitely sensitive points on the surface. These areas should be painted with liquid carbolic acid, a measure which nearly always gives relief.

*Large* leg ulcers, and those which are unable to contract because of adhesion to underlying bone, are best treated by skin-grafting. Whole-thickness skin-grafts give better end-results than Reverdin grafts, though this method is more certain of success (see Skin Grafting, under WOUNDS, TREATMENT OF). A pedicled flap is better still, and may be cut from the opposite thigh, this thigh and the affected leg being kept in apposition by plaster bandages until it is safe to divide the pedicle. Before performing such an operation, care should be taken to ascertain whether the proposed position will be tolerable for the patient. An acutely bent knee, for example, may become very irksome and painful to a middle-aged patient, and he may be unable to maintain the position long enough for a pedicle graft to be made.

Stretching of nerves going to an irritable ulcer, undermining of the edges of an adherent ulcer, or multiple radial incision of its border are procedures adopted by some surgeons, but are not recommended.

The large thin scars that cover ulcers from extensive burns, and from time to time break down, can only be treated satisfactorily by excising the ill-nourished part and covering the raw area with a sliding pedicle graft.

**Trophic ulcers** can only with difficulty be induced to heal. The perforated ulcer of the foot, for example, is dealt with by excising the margins with a narrow knife, curetting the walls of the sinus and painting it with pure carbolic. During the process of healing the ulcer must be protected from injury; this is the most difficult part of the treatment, because the ulcer is insensitive to pain.

C. A. PANNETT.

## ULCUS MOLLE

**ULCERS, CORNEAL** (*see* CORNEA, AFFECTIONS OF).

**ULCUS MOLLE** (*syn.* Chancroid, Soft Sore).—Non-syphilitic ulceration of the genitals simulating a syphilitic chancre. Unless transferred to other parts of the body by external agencies, the disease is confined to the sites of inoculation and the glands draining the affected part. These frequently suppurate.

**Etiology and pathology.**—The micro-organism commonly believed to be responsible is a short Gram-negative streptobacillus called Ducrey's bacillus, but there is fairly strong evidence to show that, in many cases, other micro-organisms, such as diphtheroid bacilli, streptococci and staphylococci, may set up an ulcerative process which closely simulates that following infection with Ducrey's bacillus. Infection usually results from sexual intercourse, and is comparatively infrequent, occurring in not more than 6 per cent. of cases of venereal infection.

**Symptomatology.**—After an incubation period which averages two or three days, but may be as short as one, or as long as seven or more, a papule appears at each site of inoculation, and quickly breaks down to an ulcer with necrotic base and overhanging, red-tipped edges. Coalescence of a number of these results in an ulcer which is characteristically irregular in contour. The ulcer is supple, buckling easy, and is painful and prone to bleed. The surrounding tissues are usually normal in appearance and consistence, in contrast with a syphilitic chancre, where they are thickened and infiltrated. In sites which are permanently moist, as beneath the prepuce and between the labia, it commonly happens that the first ulcer, or crop of such, is followed by others in the neighbourhood, owing to auto-inoculation. In these cases the daughter ulcers, which may be followed by still another crop, are usually smaller than the parent. The ulcers may heal very quickly, but they often last for six weeks, and may persist for many weeks or months.

**Complications.**—*Phimosis* may result from the accompanying inflammation of the prepuce. *Phagedæna*, in which, owing to secondary infection, the ulcer becomes black and necrotic and great destruction of tissue rapidly results, is very uncommon. The commonest complication is *suppuration of inguinal glands* on one or both sides. This frequently happens in chancroids which have apparently healed

quickly, and it is quite common to find only slight traces of the original sore in a patient presenting himself with a suppurating bubo. The glands become swollen and very painful, and the skin over them reddened. If not treated, the glands break down, and presently the abscess breaks through some point in the skin, which has by now become thin and glazed. Buboec which burst in this manner may prove most intractable, and are very occasionally followed by an ulceration which creeps over the groin and even over the upper part of the thigh.

**Diagnosis.**—Ulcus molle is distinguished with comparative ease from *syphilitic chancre* by its irregular, overhanging edges, and its suppleness, as well as by the absence of infiltration of the surrounding tissues, which contrast with the round or oval contour, shelving edge and toughness, and infiltration of surrounding tissues, which characterize the graver lesion. In the secretion of the latter, as well as in the juice of the neighbouring enlarged glands, may be found the organism of syphilis. Enlarged inguinal glands which are not painful, and the skin over which is not reddened, are almost diagnostic of syphilis, but suppuration of glands does not exclude syphilis.

It is important to remember that a lesion which commenced as a chancroid may take on syphilitic characteristics when the longer incubation period of the latter disease has elapsed. It is necessary, therefore, in all cases to keep a close look-out for this change, which is shown by the gradual rounding off of the edges of the ulcer and infiltration of the surrounding tissues. A careful watch in such cases will often be rewarded by the satisfaction of having diagnosed syphilis in a case of mixed infection before the Wassermann reaction has become positive, a matter of great moment to the prognosis of syphilis. In all cases the observation should extend to three months, during and at the end of which time the blood should be tested for the Wassermann reaction.

**Treatment.**—It is impossible to discuss the many forms of treatment advocated for chancroid, and I propose to describe only those which have served me well. The principle in all cases should be to do nothing that will interfere with drainage from the sore. Ointments are often responsible for the formation of bubo. When the sore is hidden behind a tight prepuce it is usually necessary to expose it by dorsal incision or by taking a broad V

## UMBILICAL INFECTION IN THE NEW-BORN

out of the prepuce. Some circumsise, but I prefer the V with its base at the free edge of the prepuce and its point opposite the centre of the corona glandis. In most cases the application of sublimed sulphur, which is rubbed in several times a day by the patient with the tip of a finger, is sufficient. In more severe cases, and especially when the glands are threatening to suppurate, long soaking of the parts in hot solution of eusol or boric acid, to either of which common salt has been added (a teaspoonful to half a pint), is indicated. This hypertonic solution promotes drainage and takes the burden off the over-taxed glands. The soaking is followed by the application of gauze soaked in the same solution and covered with waterproof tissue. When the glands inflame, rest in bed, with the application of antiphlogistine, followed by lint covered with ichthyol 1, ung. belladonnæ 2, to the groin, and of the hypertonic and antiseptic dressing mentioned above to the genitals, usually serves to avert suppuration. In cases where the glands have already suppurated I have found repeated aspiration of the contents through a stout, hollow needle (which is entered through healthy skin) and injection of electargol both simple and effective. An alternative procedure is to make a small opening in the lower internal pole of the abscess, evacuate the contents, and insert a small wick of gauze. This is the method of choice when the abscess shows no sign of closing down after it has been aspirated three or four times.

L. W. HARRISON.

**UMBILICAL INFECTION IN THE NEW-BORN. Etiology.**—The umbilicus may be infected at birth by contact with vaginal discharges or by want of surgical cleanliness on the part of the attendant. After birth, infection may be derived from the hands of the midwife, from a septic dressing, or possibly from the water of the bath. Contact with soiled clothes may be responsible if the navel is allowed to remain uncovered or is improperly dressed. Antenatal infection from the placenta is probably very rare. The infecting organisms are generally the staphylococci and streptococci, but other suppurative organisms, including the pneumococcus and colon bacillus, are occasional causes.

Separation of the umbilical cord may be retarded by infection, and in such cases *gangrene of the cord* is not uncommon, the cord becoming moist, swollen, and offensive, and

often greenish in colour. On its separation a ragged, sloughy ulcer may remain. A minor degree of infection may lead to *catarrhal omphalitis*, the cord on separation leaving behind a greyish granular surface, discharging a thin serous fluid.

In *umbilical ulceration* the puckered umbilical folds are red and swollen, and hide an ulcerated patch which discharges pus and may be covered with a fibrinous membrane. Occasionally the ulceration may be more extensive and involve the abdominal wall. The *suppurative form of omphalitis* may be accompanied by cellulitis. The navel is ulcerated and greatly swollen and oedematous, and freely discharges pus. The cellulitis may spread to the abdominal wall, causing a brawny, tender induration, perhaps marked by the red lines of lymphangitis. Abscess may form in the affected area. The constitutional disturbance is severe, with fever, rapid wasting, anæmia, vomiting, and diarrhoea, and a fatal septicæmia is to be expected. Occasionally the condition gradually improves.

*Gangrene of the umbilicus* is rare, and occurs in feeble, weakly infants subjected to bad hygienic surroundings. It is usually preceded by either ulceration or cellulitis, and is almost always fatal. Destruction is rapid and may lead to perforation of the abdominal wall, to fecal fistula, and to general peritonitis.

*Inflammation of the umbilical vessels* is a common accompaniment of umbilical infection, but may occur in an infant whose umbilicus is normal in appearance. *Phlebitis and periphlebitis* of the umbilical vein is generally fatal within a few days, the inflammation spreading rapidly along the vein to the liver and leading to multiple hepatic abscesses and general septicæmia. Jaundice appears and rapidly deepens until death. *Arteritis and periarteritis* are more common and less serious. The whole of one or both arteries may be affected, or the inflammatory process may be limited to a part of a vessel which may be continuous with or remote from the navel. Pus may emerge from the navel, and, by examination with a probe, may be shown to come from an artery. The thickened vessels can sometimes be felt through the abdominal wall. In many cases the condition is undetected, for there may be nothing noteworthy about the appearance of the umbilicus. Septicæmia rarely occurs, but local extension of the infection rather more commonly gives rise to pelvic abscess and general peritonitis. *Umbilical hemorrhage*, if

## UMBILICUS, MALFORMATIONS OF

not due to imperfect ligation, is generally due to sepsis.

Apart from the local affections just described, it must be remembered that the umbilicus is the chief avenue of infection in erysipelas, in tetanus, and in septicæmia and pyæmia, when these occur in the new-born.

**Treatment.**—If the umbilicus were treated from birth with the same precautions as a surgical wound, umbilical sepsis would be a very rare event. Any dressing must be sterile, and all instruments used must be boiled. When infection has occurred the septic focus must be treated along ordinary surgical lines: it should be kept covered by antiseptic dressings, and clean by antiseptic lotions; free drainage should be established, and areas of cellulitis and abscesses opened as they appear. Alcohol, caffeine and digitalis may be used to combat heart failure. When the infection becomes generalized, remedies are of little avail. Stimulants, especially alcohol, are indicated. For collapse, salines, either hypodermically or by the bowel, should be employed.

FREDERICK LANGMEAD.

## UMBILICUS, MALFORMATIONS OF.—

The umbilical region is the last portion of the ventral wall of the fœtus to close, and till late in fœtal life there can be found in the proximal end of the umbilical cord the remains of the vitello-intestinal duct and the urachus. The umbilical arteries and umbilical vein are obliterated soon after birth, becoming the obliterated hypogastric arteries and the round ligament of the liver respectively. Most of the malformations of the umbilicus result from some failure in the above processes.

The normal umbilicus is a puckered cicatrix forming a pit or depression in the centre of the anterior abdominal wall.

The following are the main malformations:

**Hernia.**—Four varieties are seen: (a) *Infantile hernia*, usually a small projection developing soon after birth owing to yielding of a weak umbilical scar. It often closes in if a support is worn, but frequently needs operation.

(b) The ordinary *umbilical hernia of adults*.

(c) *Hernia of the amnion or exomphalos*.—In this the abdominal wall fails to close and the amnion is spread out over the contents of the abdomen, which can be seen through it. Operation is usually hopeless, but in slight cases it is possible to bring the abdominal wall together.

## UNDULANT FEVER

(d) *Hernia into the umbilical cord*.—Care has to be taken not to divide any gut in the proximal part of cord.

**Fistula.**—(a) Persistence of the vitello-intestinal duct may leave a communication from the bowel to the surface of the body, forming a true *fæcal fistula*. Sometimes the intestinal end closes and leaves a mucous fistula opening at the umbilicus.

(b) Persistence of the urachus may lead to a *urinary fistula* at the umbilicus. The distal end of the duct may form a fistula not communicating with the bladder.

These fistulæ must be distinguished from acquired fistulæ. Such are biliary fistulæ, which may form after inflammation of the gall-bladder due to gall-stones, and serous fistula, which may develop in cases of tuberculous peritonitis and from which large quantities of ascitic fluid may pour away. Fæcal fistula may also develop at this site in tuberculous peritonitis.

**Cysts** may form near the umbilicus, appearing as swellings of varied size, either subcutaneous or subperitoneal. They are formed from unobliterated parts of the urachus or omphalomesenteric duct.

**Granuloma** of the umbilicus is a small red swelling forming soon after birth. It is easily removed by scissors or ligature.

A **polyp** forming a small red swelling may develop from remnants of the vitello-intestinal duct.

Concretions of epithelial debris and dirt sometimes form in the umbilical depression. Ulceration and suppuration may occur.

Secondary carcinoma not infrequently occurs at the umbilicus. It may follow carcinoma of the stomach, gall-bladder, uterus, or ovaries.

ZACHARY COPE.

## UNCINARIASIS (see INTESTINAL WORMS).

**UNDULANT FEVER** (*syn.* Malta Fever, Mediterranean Fever, Rock Fever, etc.).—A pyrexial disease due to infection with *Micrococcus melitensis* or *paraneitensis* (PLATE 1, Figs. 7, 8, Vol. I, facing p. 146), and characterized by febrile manifestations of long and uncertain duration and irregular course with a marked tendency to undulatory pyrexial relapses.

**Etiology.**—The *Micrococcus melitensis* was discovered by Sir David Bruce in 1886, and was isolated by him from the spleen of a fatal case occurring in Malta. The means by which



## UNDULANT FEVER

the organism gains entrance into the body were, however, unknown until 1904, when, as a result of the work of the Malta Fever Commission, it was discovered that goats suffered from undulant fever, that the urine and milk of affected goats contained the micrococcus, and that it was by means of infected goat's milk that the disease was conveyed to man. The alimentary canal is for all practical purposes the only channel by which this micrococcus enters the body.

**Geographical distribution.**—The endemic area of the disease is the Mediterranean, its shores and islands. It was formerly very prevalent in the Navy and Army in Malta and Gibraltar, until it was practically suppressed by prophylactic measures on the lines indicated by the etiology. Undulant fever occurs also in Greece, Italy, Spain, the South of France, Portugal, India, South Africa, Ceylon, and, in fact, in all parts of the world where goats are kept and goat's milk is used for human consumption; but it is most common in tropical and subtropical climates. Undoubted cases of the disease have occurred in England.

**Pathology.**—The micrococcus enters the blood through the mucosa of the alimentary canal and causes a septicæmia, but without producing any lesions in the alimentary canal itself. Agglutinins, which may persist for years, are formed in the blood. The spleen is enlarged and congested, as are the liver, kidneys, mesenteric glands, small and large intestines, and lungs. The tissue of the spleen is dark red and friable, and the Malpighian bodies are enlarged. The kidneys sometimes show a glomerular nephritis, and patches of consolidation may be met with in the lungs. The brain is generally congested and the heart-muscle shows degeneration. The micrococcus may be isolated from the blood, spleen, and internal organs.

**Symptomatology.**—The *incubation period* is difficult to fix exactly in a disease with such an insidious onset, but as a rule it is fourteen days. The symptoms are very variable, but may be described as they occur in three main forms—(1) the malignant or fulminating form, (2) the intermittent form, often mild, (3) the undulant form, which is the commonest and gives rise to the name of the disease.

(1) **Malignant or fulminating form.**—In most cases the onset is sudden, and attended by severe headache, "pains all over," anorexia, nausea or actual vomiting, flushed face and dry skin. The temperature rises rapidly, and

will be found to be 104° F., or even higher, within the first twenty-four hours. The patient may complain of epigastric and splenic or hepatic pain, and there will be tenderness in these regions on palpation. The tongue is furred but clean at the tip and edges, the breath offensive, and diarrhoea may be present; if so, the stools are copious, loose, offensive and bile-stained. The pulse-rate is raised, the respirations are increased, and there are often signs of hypostatic congestion at the bases of the lungs. The spleen will be found enlarged. The urine is scanty, high-coloured, and shows a pronounced uratic deposit. The temperature usually remains high for four or five days, when there may be a remission, during which the range is lower, the evening temperature rising to about 102° F. The fever continues at this lower level, and after a varying number of days—generally five or six—the patient shows signs of increasing exhaustion, the heart begins to flag, the pulse becomes feeble and intermittent, the respiration more laboured and obstinate, and vomiting may now set in. An offensive odour may emanate from the skin and breath. Pulmonary congestion becomes more marked and the respirations more embarrassed, delirium supervenes, and fæces are passed involuntarily. The temperature rises gradually or suddenly, coma follows the delirium, and the patient dies from cardiac failure or hyperpyrexia, his whole appearance suggesting a condition of toxæmia.

In some cases, when the remission of temperature occurs the patient falls into a state of nervous excitability, followed by extreme nervous exhaustion, ending fatally. In other cases the primary wave of pyrexia may be safely passed through, but the disease proves fatal during the secondary wave of pyrexia, even after a lapse of some weeks. The malignant form of undulant fever is fortunately comparatively rare.

(2) **Intermittent form.**—The disease usually commences insidiously, the general course is mild and the duration less than in the undulant type. First there is gradual loss of appetite, with malaise and headache, and with a slight rise of temperature in the evening. The symptoms increase in severity, the headache becomes worse, there are pains in the back and neck, and sleeplessness and evening perspirations. The tongue is furred, and some epigastric discomfort and tenderness are present. The morning temperature will now be found to be about 100° F. and the evening

## UNDULANT FEVER

temperature 101° F. This high temperature may continue for six weeks, two months, or even longer. The patient usually feels comparatively well in the earlier part of the day, but in the afternoon, about 2 p.m., there is a sensation of chilliness and the temperature rises to 102° F. or 103° F. beginning to fall about 6 p.m., accompanied by sweating.

No other symptoms may be present, and for this reason the real nature of the fever may for some time be overlooked. Increased debility and anæmia, however, characterize the later stages of the fever. The intermittent type of fever may be maintained throughout the course of the disease, the temperature gradually falling, or passing into the undulant type. It may also occur at the termination of an undulant attack.

(3) **Undulant form.**—This, the most common and characteristic form of the fever, is marked by undulations of a remittent pyrexia of variable length separated from each other by apyrexia and temporary absence of symptoms. The fever begins gradually, the temperature remitting each morning about half the previous evening rise. During this time the patient complains of headache and pains in the back and limbs, and there are epigastric tenderness, a furred tongue, and constipation. In about a week or ten days the temperature is found to be 103–105° F. at night, and all the symptoms are aggravated, particularly the headache, which is usually severe. At this time there is evidence of some bronchial catarrh or hypostatic congestion of the lungs. The temperature now begins to decline, and in a few days' time registers only 99° F. or normal in the morning, although it is still slightly raised at night. As it falls the patient feels much better, all the symptoms ameliorate, and the primary wave of the fever is at an end. The temperature remains normal throughout the twenty-four hours, or is slightly raised at night, for a few days, when it begins to rise again, and all the symptoms are repeated, although perhaps not in so severe a form as in the primary fever. This secondary undulation subsides in its turn, and again there is an interval of actual or relative apyrexia, to be followed by other undulations of fever. This condition of alternate waves of fever and apyrexial intervals may last for months, and sometimes for as long as eighteen months or two years. During the later febrile attacks most profuse perspirations attend the daily rises of temperature, and the patient is obstin-

ately constipated. As the disease progresses, muscular wasting and anæmia become very pronounced, and neuralgias of various kinds occur. Sudden painful effusions into the joints, rheumatic pains in the limbs, and orchitis are of common occurrence. The patient, in a fully developed case of the undulant form, presents a characteristic picture: he is thin, wasted and anæmic, suffers from a remittent fever attended with copious daily sweating, and possibly from swollen knee-, ankle-, or wrist-joint, and is the subject of severe neuralgias and attacks of bronchial catarrh. He is depressed and miserable, and to add to his sufferings, resulting from the profuse sweats, sudamina are common, and in the hot weather he may be covered with prickly-heat lesions.

Some of the more usual symptoms and physical signs met with in undulant attacks must now be described in a little more detail.

The pulse-rate is usually slow and full at the beginning of the fever, but as the disease progresses becomes more rapid, and in long-continued cases is constantly high—110 to 120 per minute. The heart's action becomes feeble, palpitation occurs on slight exertion, and hæmic murmurs may be heard on auscultation. The tongue is always furred at first and in the pyrexial waves, but cleans in the intervals, when appetite returns. In much-protracted cases the tongue becomes glazed and red; it does not finally clean until the temperature becomes subnormal.

The liver may be slightly enlarged, and is often tender on pressure. The spleen is always enlarged, especially in the malignant form, and is tender at first; it decreases in size in the later stages of the disease. Bronchial catarrh is usually present during the pyrexial periods, and there is often basal congestion of the lungs. Neuralgia may occur at any stage of the disease, but not usually during the primary wave of pyrexia. It may be frontal, occipital, or facial, or may take the form of lumbago or sciatica.

Effusions into the joints may be acute or subacute, the former occurring during the pyrexial attacks and suddenly affecting the larger joints, as the knee, hip, or shoulder, and the latter attacking the smaller joints, as the wrist, and passing to the fingers, or from the ankles to the toes. The condition of the joints resembles that of an acute non-suppurative synovitis, and is extremely painful while it lasts; the acute attacks subside in a few

## UNDULANT FEVER

days, but the subacute may persist for some weeks. Complete recovery is the rule.

A mild case of the undulant form lasts two months, more severe cases eighteen months to two years. Fortunately the mortality of this variety of the fever is only some 2-3 per cent., and therefore, after a time, at the termination of a pyrexial undulation, the type of temperature becomes that of a gradually decreasing intermittent fever of small range followed by an absolute return to normal or subnormal temperature both night and morning. Concurrently with the fall of temperature there is a mitigation of all the symptoms; the patient now begins to improve in all respects, the tongue cleans and the appetite returns. When convalescence is thoroughly established, he gains in weight and strength, although complete restoration of health may be somewhat prolonged.

**Blood changes.**—There is a secondary anemia in the later stages of the disease, and a decrease in the red blood-cells of 20-40 per cent. There is no great change in the total number of white blood-corpuscles, although a differential count may show a decrease in the relative proportion of the polymorphonuclear cells and an increase in the large mononuclears.

**Complications.**—In addition to those already mentioned as frequently occurring in the course of the disease, such as the joint-effusions, there may be pericardial and pleural effusions, meningitis, an acute condition of cerebral irritability, and local abscesses.

**Diagnosis.**—The diagnosis may be impossible, especially in the early stages and in the malignant form, without the aid of bacteriological methods. Undulant fever is not infrequently mistaken for *enteric*; bacteriological diagnosis by means of blood-cultures, by isolation of the micrococcus from the urine and by agglutination tests, should always be carried out. In the later stages, particularly in the undulant form, the character of the temperature chart, the profuse sweating, the joint-affection, and the neuralgias which are so frequently present, should serve to make the diagnosis plain.

Differential diagnosis may have to be made from fevers of the enteric group, as already mentioned, by bacteriological methods and the general symptoms; from *malaria* by the presence of the parasite in the blood in the latter disease; from *septicæmia* by the blood picture and different types of temperature, and possibly from *kala-azar* by the blood picture,

## URETHRA, PROLAPSE OF

the temperature chart, and the greater splenic enlargement in the latter disease.

**Treatment.**—There is no specific drug treatment. Serum treatment has not fulfilled expectations, but vaccine treatment, as recommended by Bassett-Smith, either with ordinary autogenous or sensitized autogenous vaccines, should be tried. The best results have been obtained when the vaccine has been given during the apyrexial intervals. It may be injected in doses of 50-200 millions once a week, or in smaller doses of 10 millions at more frequent intervals. Yeast in 2-dr. doses has also been tried, and favourable results have been reported. Nursing is of great importance, and the patient should be clothed in light flannel and especial care taken to avoid chills, although good ventilation and plenty of fresh air are important. Symptomatic treatment will also be required, and a careful watch should be kept on the heart and lungs. During the fever periods a fluid diet is indicated, but during the intermissions of fever and in convalescence, light but nourishing food should be given. Alcohol is generally necessary during the course of the disease. A change to a temperate climate, except during the winter, is usually necessary after recovery. The careful disinfection of all excretions, faeces, saliva, and urine, especially the urine, should be a routine measure.

OLIVER ROBINSON.

**URÆMIA** (*see* NEPHRITIS).

**URETERIC CALCULUS** (*see* URINARY CALCULI).

**URETHRA, PROLAPSE OF.**—Prolapse of the female urethra is found in debilitated children (representing 50 per cent. of the cases) and in women over 40 (40 per cent.). The cause is unknown, but loss of tone in the circular muscle-fibres permits dilatation and stretching of the mucous membrane, which comes to glide upon the submucosa. Calculus, straining, cystitis, and parturient trauma predispose. The **symptoms** are slight bleeding, frequent micturition, pain, and a dark, congested swelling, which occupies the site of the meatus, and in which the urethral canal is found. **Treatment** is operative. The prolapsed mass may be simply excised, but a better procedure is, with a staff in the urethra, to replace the lining and cut down upon the urethra throughout its length; the muscle-layer is then plicated so as to reduce the diameter of the urethra throughout its length.

BRYDEN GLENDINING.

## URETHRAL CARUNCLE

**URETHRA, RUPTURE OF** (*see* URETHRAL STRICTURE).

**URETHRAL CALCULI** (*see* URINARY CALCULI).

**URETHRAL CARUNCLE.**—Urethral caruncles are commonly seen on the posterior wall of the female urethra, usually at the margin of the meatus. The cause is unknown. Either a mild urethritis or a chronic leucorrhœa is commonly associated, and may be looked upon as a predisposing factor. The condition is comparatively often seen in patients of 40-60, but may occur at any age. Both the married and the unmarried are affected, but the latter less commonly, and, on the average, at a later age-period.

**Symptomatology and pathology.**—Of the symptoms, pain is the most constant, though varying greatly in intensity and duration. It is often brought on by walking or other action, and is a common cause of dyspareunia in middle life. Sometimes micturition is so painful as to lead to retention. Hemorrhage is a trivial but fairly constant symptom. The bleeding does not, as a rule, amount to more than a drop or two of blood lost irregularly either at night or during the day. The blood may be passed at the end of micturition with the last few drops of urine.

The caruncle forms a bright-red tumour, seldom larger than a pea, appearing at the posterior or the lateral lip of the meatus, and perhaps extending for some distance up the posterior wall of the urethra. One variety forms an ill-defined red patch, scarcely raised above the surrounding mucous membrane, sessile, rounded, of a pale-red colour, and often insensitive to the touch. Microscopically, this type consists of a vascular inflammatory round-cell infiltration beneath a stratified epithelium which appears thinned and stretched over it. It would appear to be a true granuloma, and is associated with a urethritis. A second variety, which is equally common, forms a bright-red raised mass, either pedunculated or finely lobulated, and in many cases very sensitive when touched. Microscopically, it is in the nature of a very vascular papilloma. The epithelium is of the stratified squamous variety, arranged in flat-topped elevations with intervening sulci, resting upon a very vascular connective tissue, deep to which are found round-cell inflammatory products. Sometimes the vascularity is such as to suggest an angioma.

**Treatment.**—The form of treatment which

## URETHRAL STRICTURE

gives the best results is free excision under general anæsthesia and careful suturing of the wound. If necessary, the urethra should be dilated, or even split and re-sutured in order to make sure that the caruncle is completely removed. Unfortunately, these cases are seldom admitted to the wards of a surgical hospital, and recourse is had to cocaine (10 per cent. solution) of the meatus in the out-patient department, and application of the actual or electric cautery. Recurrences are fairly frequent (estimated at about 50 per cent. within two years) by this method, chiefly because it is, under these conditions, often impossible to burn the upper part of the growth in the canal. Better results are obtained with a general anæsthetic, dilatation of the urethra, and thorough cauterization.

BRYDEN (GLENDING).

**URETHRAL STRICTURE.** Stricture of the urethra may be congenital or acquired. **Congenital** strictures are usually situated at, or immediately behind, the external meatus. A less common site is in the bulb or the bulbomembranous junction. They may be cylindrical (when a considerable length of the urethra is involved), annular or diaphragmatic (when the constriction is linear), or bridle (when one wall of the urethra is raised up in the form of a crest). The same symptoms may be caused by a simple fold of the mucous membrane which acts as a valve. The bladder may be hypertrophied and the kidneys and ureters dilated at birth, but this is very rare. Phimosis usually accompanies meatal strictures. The **symptoms** are difficulty of micturition and, often, pain during the act. Incontinence of urine in boys, when it is both diurnal and nocturnal, is occasionally due to a congenital stricture in the deep urethra. **Treatment:** As it is impossible to dilate meatal strictures, they should be divided (meatotomy); indeed, in all cases in which a circumcision is performed, the terminal portion of the urethra should be examined to ascertain its calibre, and when necessary a meatotomy performed. Congenital strictures of the deep urethra can easily be dilated.

**Acquired** strictures are inflammatory or traumatic.

### INFLAMMATORY STRICTURE

Over 95 per cent. of inflammatory strictures are due to gonorrhœa. Tubercle, syphilis, and the urethritis which very occasionally

## URETHRAL STRICTURE

follows the acute infective fevers may give rise to stricture. A moderate degree of urethral stricture is usually present in men who have been on catheter life for a considerable number of years. In such cases it is due to a mixture of inflammation and trauma.

**Site.**—The most common site is just in front of the bulb, in which position the first evidences of stricture formation are found. Secondary strictures are very frequently encountered in front of this, the most common sites being at the angle between the penis and scrotum, and just inside the meatus. Frequently four, five, or even more strictures are present, and if a sound is placed in the urethra they may be felt as hard ridges or rings around it, on external palpation. In cases of multiple stricture, that in the bulb is always the tightest but usually the most easily dilated. Gonorrhoeal stricture of the posterior urethra is almost unknown.

**Time of onset.** Hospital patients usually present themselves for treatment ten to fifteen years after infection by the gonococcus, but as a rule private patients are seen much earlier. As the vast majority of cases of gonorrhoea occur between the ages of 20 and 30, it follows that stricture is most commonly noted in the fourth and fifth decades.

**Pathology.** Stricture must be considered as the possible ultimate stage of every chronic urethritis. It is essentially an inflammatory sclerosis, and tends to progress as long as the inflammation is allowed to persist. As the calibre of the urethra is usually reduced to a fraction of the normal before the patient seeks treatment, the earlier stages of stricture formation are only to be observed in a routine urethroscopic examination of patients suffering from chronic urethritis. In these cases there is a definite infiltration of the submucous coat by fibrous tissue, chiefly to be found round the mouths of the infected glands and follicles. It is most evident in the three definite localities mentioned above, while the process is always more advanced just in front of the bulb than elsewhere. If the urethra is examined under air-distension, definite sickle-shaped folds of mucous membrane are to be seen projecting into the lumen; they are due to the contraction of the underlying fibrous tissue, and give the appearance of a thin white thread stretched beneath the mucous membrane. These folds usually extend about half-way round the circumference of the tube, but occasionally they form a complete ring.

If the fold involves only a portion of the circumference, a second usually forms on the opposite side of the tube, either just in front of or behind the first. As the process of cicatrization advances, a composite stricture is formed by several of these folds coalescing into one long stricture. This explains why the opening of the stricture is usually eccentric, and the narrowed part of the urethra tortuous. If the condition is untreated, the stricture tends to become both narrower and longer. The stricture itself is formed of a dense mass of fibrous tissue, the fibres of which run for the most part circularly round the urethra.

**Symptoms.** The first symptom is a slight chronic urethral discharge, which may be noticed only in the morning; it is thin, white in colour, does not cause any inconvenience, and is usually dismissed by the patient as "only a gleet." If it remains unnoticed or ignored, he presents himself for examination when the act of micturition is definitely altered. The stream is then poor and weak, the jet forked, or twisted, or coming as a spray; in extreme cases the urine only comes drop by drop. Most of the alterations in the nature of the jet are due to the shape of the external meatus, and to the fact that, owing to the obstruction, the stream of urine is not strong enough to distend the urethra in front of the stricture. Loss of force of the jet is due to the same cause, and the patient finds that he is unable to project the stream away from him, the urine splashing down over his boots. Straining increases both the volume and the force of the stream. Micturition is often interrupted, the stream dying away slowly, only to be restarted in a few moments. This is due to fatigue of the bladder muscle, which is unable to force the urine out in one sustained effort. "After-dribbling" is a rule in cases of tight stricture; it is due to the small amount of urine which has collected in the dilated urethra behind the stricture leaking out and soiling the patient's clothes after each act of micturition. If there is cystitis, frequency and precipitancy of micturition are also present. The urine may be clear, but it always contains threads and flakes (evidence of urethritis), and if the bladder or kidneys are infected it is uniformly turbid from pus. There is generally a scalding or burning *pain* felt along the urethra during micturition. If there is cystitis or a partial retention, a suprapubic ache is felt. Pain in

## URETHRAL STRICTURE

the renal regions is a serious sign; it is caused either by a pyelitis or by back pressure on the kidneys. *Acute retention of urine* is the initial symptom in about 7 per cent. of cases, but is much more common after rough and injudicious attempts to dilate the stricture; it is due to congestion caused by alcohol, constipation, etc., or to reactionary swelling after the trauma inflicted by the instrument. *Hæmaturia* is very rarely a symptom of stricture; if it occurs apart from instrumentation a cystoscopy should be performed as soon as possible, to exclude bladder growth, etc.

**Complications** are partly mechanical and partly inflammatory. The mechanical effects of a stricture consist in the dilatation of the whole urinary tract above it. For a time the bladder hypertrophies, but in the later stages its muscle is unable to overcome the resistance, and the bladder becomes distended. The inflammatory complications are prostatitis, epididymitis, cystitis, pyelitis and pyelonephritis, all of which are considered elsewhere, as also are extravasation of urine and periurethral abscess. Accidents in treatment and urinary fistula are included in this article.

**Urinary fistula** is usually the result of a periurethral abscess that has been allowed to burst, or has only been opened when pointing. If the fistula is short and straight it does no harm to the general health, though it causes great discomfort, as some of the urine comes through it at each act of micturition. Occasionally, more especially if the stricture is tight, the fistula tracks in different directions under the skin, and attains the surface by many openings. There may be a considerable increase of fibrous inflammatory tissue round the openings, and this often gives rise to a hard, irregular, nodular tumour in the perineum, which is studded with fistulæ, through which thin watery pus and urine exude. The condition is called a "chronic extravasation," and has often been mistaken for a malignant growth.

**Diagnosis.** In every case of suspected stricture the patient should be asked to pass water in the presence of the practitioner. If the urine only comes drop by drop the stricture is very tight, and nothing but a filiform bougie will pass; if the stream is continuous but thin and weak, its calibre will probably be below 10 (French scale); on the other hand, a good flow of urine does not preclude the presence of a stricture. Urethroscopy brings to light many unsuspected strictures,

but in longstanding cases the secondary contractions prevent the tube of the urethroscope from passing down to the main stricture, so that comparatively little information is obtained by this means. Continental surgeons recommend olivary bougies for the purpose of diagnosis. They have a large head and a very slender shaft, and give accurate information, as the sensation when the head slips through a stricture with a jump is very characteristic. However, the calibre of a stricture can easily be measured by means of ordinary soft gum-elastic bougies; these should be calibrated according to the French scale, as there is too big a gap between consecutive sizes in the English scale. If a stricture is suspected, a medium-sized instrument—about No. 18 French—should be tried. If it does not pass, substitute one about six sizes smaller, and so on till a bougie is found that passes readily into the bladder; then increase the size of the bougie until one is gripped by the stricture. This gripping of the instrument is very characteristic, and "is the essential diagnostic sign of stricture." For example, if Nos. 18 and 12 fail to pass, but Nos. 6 and 7 pass easily, and No. 8 is gripped, we know that the calibre of the stricture is 8 F. This is sufficient information for one sitting, and the patient should be asked to return in a week. At the second sitting Nos. 7, 8, and 9 should be passed, and at the third, Nos. 8, 9, and 10, and so on. It is always better to begin a sitting with an instrument one size smaller than the largest passed at the previous visit, and it is a mistake to endeavour to dilate the stricture by more than one size at a sitting. If no instrument can be passed, it means either that there is an impassable stricture or that there is none at all. If there is any bleeding after these failures, it probably means that a very tight stricture is present, in which case it is better to postpone further attempts to pass instruments for a few days. If there is little or no bleeding, the largest steel instrument that will pass the meatus should be tried. Usually by exercising patience and gentleness it will be possible to insinuate a fine instrument through any stricture which permits urine to pass. Finally, if no instrument will pass, the patient should be anesthetized and a last attempt made. This failing, a Wheelhouse operation should be performed at once.

**Treatment.** (1) **Preventive.** This consists in the careful treatment of gonorrhœa (q.v.).

(2) **Dilatation** is essentially the treatment of

## URETHRAL STRICTURE

all strictures, and must be done methodically, no matter whether an operation is performed or not. It should always be carried out by the practitioner; except in very special circumstances, it is a great mistake to allow a patient to dilate his own stricture. All instruments should be passed with extreme gentleness and deliberation.

(a) *Intermittent dilatation*.—This consists in passing instruments at regular intervals. Below 18 F., soft gum-elastic bougies should be used; above this size, steel sounds are better. At the first sitting it is enough to estimate the calibre of the stricture. At the second, three instruments should be passed—the size below the largest passed previously, and the next two sizes. Thus the stricture is dilated one size at each sitting. When soft instruments are used the interval between each sitting should be a week; with steel bougies a fortnight's interval is better. The stricture having been dilated to 18 F., it is best to start with an 8/10 steel sound, and continue with these instruments up to 14/16. When the stricture is fully dilated the intervals between the sittings should be increased until the maximum interval without recontraction has been reached. For example, if there is no recontraction after a three-months' interval, but there is after a four-months', three months is the patient's maximum interval, and he should have instruments passed every three months for the rest of his life. If the maximum interval is a year or longer, the patient may be discharged safely, as there is then no risk of recontraction; but this only occurs in a few cases.

(b) *Continuous dilatation*. This method is most useful in tight strictures. If a filiform bougie has been passed only with difficulty, it should be tied in place and the patient put to bed; the urine will dribble away beside the bougie. The next day it will be found that an instrument about two sizes larger can be passed; this also should be tied in. After another twenty-four hours the instrument may be removed, and the patient will be able to pass urine easily. Further treatment is by intermittent dilatation.

(c) *Rapid dilatation* is only mentioned to be condemned. The patient is anæsthetized, and a large number of instruments are passed until the full size is reached. But the stricture is not dilated—it is merely split, and it rapidly recontracts, and becomes much more intractable than before.

*Accidents during instrumentation.* (i) *Hæmorrhage*.—It should be the aim of the operator to avoid bleeding, but in many cases the mucous membrane of the urethra bleeds almost at the slightest touch. As in such cases there is always a considerable degree of urethritis, the urethra should be irrigated beforehand with oxycyanide of mercury (1 : 10,000). The sitting should be terminated at the beginning of the bleeding. Usually it stops in a few minutes, but if it is severe the patient should be put to bed and ice applied to the perineum.

(ii) *False passage*.—With care, gentleness, and suitable instruments this accident should not occur; it is to be suspected if the instrument, after meeting with a resistance, suddenly jerks forwards. The patient experiences a sudden sharp pain, and blood immediately appears at the meatus. The surgeon feels a slight grating transmitted up through the instrument as its point ploughs its way through the tissues. If the bleeding is at all severe the patient should be put to bed and kept under observation. Even in slight cases no instrument should be passed for a fortnight, and then it is best to commence with one three or four sizes smaller than that with which the false passage was made. The surgeon should aim at dilating the stricture with the minimum amount of damage, as trauma always predisposes to the formation of fresh fibrous tissue in the wall of the urethra, and so makes the stricture denser and tougher. For this reason I never use a local anæsthetic in the treatment of stricture, as I consider that if a dilatation is carried to the point of producing pain it probably does more harm than good.

(iii) *Catheter fever*.—In this condition the patient is seized with a rigor a couple of hours after instrumentation, and his temperature may rise to 104° F. or even higher. Urinary infection is the predominant factor, but rough and excessive instrumentation, and trauma, are predisposing factors. The treatment consists in putting the patient to bed with plenty of blankets, hot bottles, etc., clearing out his bowels by means of an enema, and giving him plenty of hot drinks. A draught containing quinine sulphate 10 gr. is also beneficial. If a patient is known to be liable to catheter fever he should be put to bed before an attempt at instrumentation is made and given a brisk purge. After the dilatation, quinine and hot drinks should be administered, and he should

## URETHRAL STRICTURE

not be allowed up for at least twenty-four hours.

Strict asepsis during instrumentation should be observed (*see* CATHETERIZATION), and if urethritis is present the urethra should be washed out with oxycyanide of mercury (1:10,000) beforehand. Lavage of the urethra after instrumentation is advisable, but in many cases it causes pain. The patient should take hexamine or some other urinary antiseptic for about three days before and after each sitting.

(3) **Operative treatment.**—If no instrument can be passed that is, if the stricture is "impassable"—a Wheelhouse operation is indicated. Internal urethrotomy is appropriate if it is impossible to dilate a narrow stricture, or if it is necessary to dilate it rapidly. External urethrotomy is chiefly indicated in cases where perineal abscess, fistula, or extravasation of urine is present. A fistula and all the inflammatory tissue around it should be excised.

### TRAUMATIC STRICTURE

This is the inevitable outcome of a rupture of the urethra, but its severity depends largely on the treatment received at the time of the injury. If the patient was operated on immediately after the accident, and the edges of the divided urethra were accurately sutured together, the resulting stricture is rarely severe enough to cause symptoms, and may not even need instrumentation. If, on the other hand, expectant treatment was carried out at the time of the accident, the stricture is exceedingly dense and hard, and often impossible to dilate. Traumatic stricture may occur either in the anterior or posterior urethra.

*Rupture of the posterior urethra* is due (a) to a severe injury involving fracture of the pelvis, or (b) to a blow on the perineum without fracture. In the first case the urethra is wounded by a fragment of bone; in the second it is torn at its attachment to the triangular ligament.

In fracture of the pelvis it is difficult to distinguish between a ruptured urethra and an extraperitoneal rupture of the bladder. If the urethra is torn it leads to bleeding from the meatus apart from micturition, there is retention of urine with distension of the bladder, and it is impossible to pass a catheter; while if the bladder is ruptured there is no urethrorrhagia, no distension of the bladder, and a catheter passes easily. In doubtful cases a suprapubic cystotomy should be done first,

## URETHRITIS

and then the examination completed. The torn urethra can afterwards be sutured through a perineal incision.

When the rupture is due to a blow on the perineum, the point of impact is far back near the anus, and the direction of the force is from behind forwards. The symptoms are those mentioned above. The treatment is to suture the urethra and institute suprapubic drainage of urine.

*Rupture of the anterior urethra* is usually caused by a blow on the perineum, but in this case the force is directed from before backwards. The symptoms are pain, urethrorrhagia, perineal hematoma, and retention of urine; and treatment is by suture of the urethra and deviation of the urine by means of a suprapubic cystotomy.

If these indications are carried out, the resulting stricture will not be severe and can easily be kept dilated. If a dense stricture forms, its onset is very rapid, and the stenosis is considerable within a few weeks. It is always single, and most evident on the floor of the urethra. It gives rise to the same symptoms, physical signs, and complications as inflammatory strictures. If possible, the stricture should be dilated, but if this method is insufficient to keep it open it should be resected, and the divided ends of the urethra sutured together. Suprapubic deviation of the urine is essential until the wound has healed.

† SWIFT JULY.

### URETHRITIS, GONORRHOEAL (*see* GONORRHOEA).

#### URETHRITIS (Non-gonorrhoeal). **Etiology.**

—Inflammation of the urethra not due to gonococci may be transmitted to a male from a female suffering from leucorrhoea. It may follow intra-urethral instrumentation or the use of concentrated urethral injections. It accompanies a stricture or a calculus impacted in the urethra. It may be secondary to a chronic prostatitis or vesiculitis of haematogenous origin, or that which remains when a gonorrhoeal infection has died out. An ingestive urethritis sometimes follows the consumption of certain articles of diet such as rhubarb or asparagus, or the taking of certain drugs such as turpentine, cubeba, copaiba, cantharides, arsenic, or potassium iodide. In some eruptive fevers—in measles, for example—it occurs rather uncommonly. Gout is an occasional cause. An unusual origin is herpes of the urethral mucosa.



## URETHROSCOPY

The infecting organism may be the *Bacillus coli*, the staphylococcus, the *Micrococcus catarrhalis* or diphtheroid organisms. The tubercle bacillus may cause a discharge when there is a lesion in the prostate, and such a discharge may be a concomitant of a tuberculous epididymitis.

The **signs and symptoms** are similar to those of the gonorrhoeal infection (*see* GONORRHOEA). But a mild brief course is usually followed, and the incubation period is more apt to be short. The discharge may never pass beyond the mucoid stage, and, indeed, does not usually do so in ingestive urethritis, in gout, mumps, or measles. The urethritis of herpes is very painful, and is preceded or followed by herpetic vesicles externally.

illumination" consist of a straight tube fitted with an obturator. This is passed down the urethra, and the obturator is withdrawn. The handle of the instrument is then attached to the outer end of the tube. It carries a small electric lamp, fixed on a long stalk, so arranged that when the handle is in position the lamp is less than a quarter of an inch from the inner end of the tube. The observer looks down the tube past the lamp, and can observe the whole length of the urethra as the instrument is slowly withdrawn. This instrument has the advantage of giving a good illumination of the portion of the urethra immediately beyond the end of the tube, but the lamps are delicate, expensive, and only last for a very

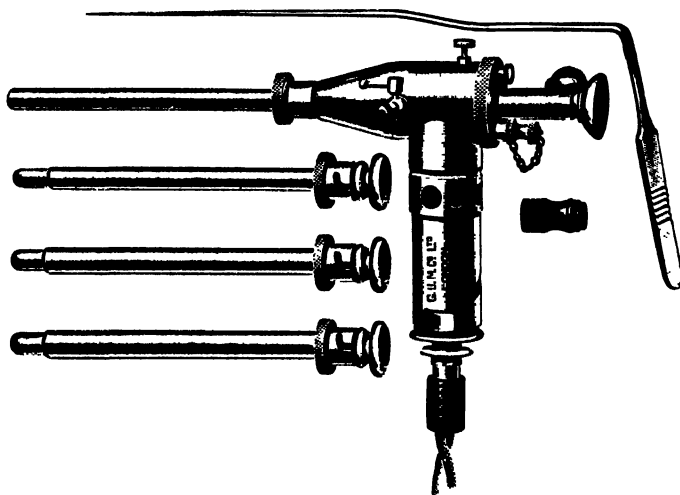


Fig. 100.—Anterior urethroscope (author's pattern).

**Treatment** follows the general lines laid down in gonorrhoea. The milder forms call for no local measures. Permanganate injections are not so efficacious as solutions of the silver salts and oxycyanide of mercury.

C. A. PANNETT.

**URETHROSCOPY.** The ocular examination of the interior of the urethra is subdivided into anterior and posterior urethroscopy, according to whether the anterior or the posterior urethra is examined, the reason being that it is necessary to use a different type of instrument to obtain the best result in the one case or the other.

**Anterior urethroscopy.**—Urethroscopes are of two types, according to the position of the source of light. Instruments of "internal

short time. In addition, it has the disadvantage that it is not suitable for use under "air-distension." The Luys urethroscope is the best-known instrument of this type.

In instruments of "external illumination," which are more commonly used in this country, the electric lamp is placed in the handle, and its light is reflected down the urethra by means of a mirror. The observer looks through a small hole in the centre of the mirror. An air-bellows is attached to a side tube, so that the urethra can be distended with air during the examination. This is a great advantage, as it enables a comprehensive survey of a large portion of the urethra to be made without moving the tube. The best-known instruments of this type are Wyndham Powell's and the author's (Fig. 100).

## URETHROSCOPY

**Technique of anterior urethroscopy.**—A tube is selected that comfortably fits the meatus. It is lubricated and passed down the urethra. The obturator is withdrawn, and the inside of the tube is mopped out by means of a small wisp of cotton-wool on a wooden or metal holder. This is to remove excess of lubricant or water that may be adhering to it. The urethroscope is attached and the light switched on. Air is admitted into the urethra from the bellows, which should previously have been blown up, and as it enters, the walls of the urethra will be observed to separate. The air forms a cushion between the urethral wall and the urethroscope tube, so that the latter can be moved freely up and down, or from side

rare conditions of the urethra. (2) *Operative.*—Infected follicles of Morgagni, small peri-urethral abscesses, and cysts of Littre's glands can be opened and cauterized, warts and polypi removed by means of a curette or by diathermy, foreign bodies extracted, and topical applications brought into accurate contact with the diseased portion of the urethral wall.

**Posterior urethroscopy.**—Any anterior urethroscope can be used in the examination of the posterior urethra, provided a long tube is used, but much better effects are to be gained by using a special instrument with a telescope resembling that of a cystoscope. Practically all modern posterior urethroscopes are designed to be used under water-distension, but in a

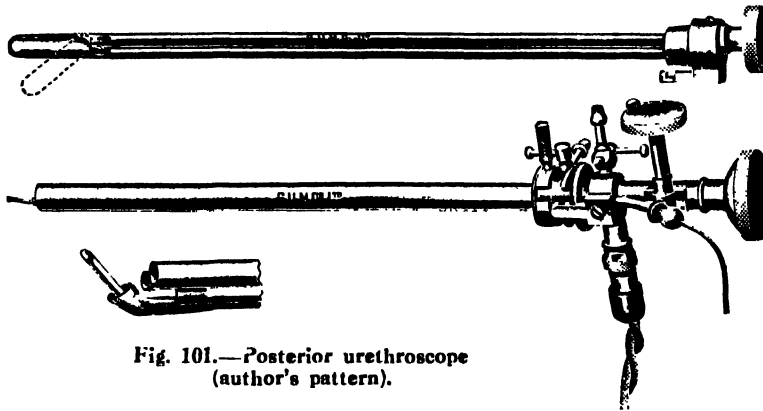


Fig. 101.—Posterior urethroscope (author's pattern).

to side, without damaging the mucosa or hurting the patient. It is best to examine the bulb first, and then the rest of the urethra from behind forwards, but if the canal is distended the urethroscope tube can be passed downwards to re-examine the urethra without re-inserting the obturator. It is important to use as large a tube as possible, as with small tubes not only is the view restricted, but the air quickly escapes at the meatus. It is possible with modern instruments to operate on the urethra under air-distension.

**Indications for anterior urethroscopy.** (1) *Diagnostic.*—Urethroscopy is essential for the diagnosis of the nature and site of the lesions in chronic anterior urethritis, and for the detection of small papillomata or polypi of the urethra. It is most useful in the diagnosis of stricture, especially in the early stages, as it enables the surgeon to determine at once the number, positions, and calibres of the various contractions. Urethroscopy is necessary for the diagnosis of pouches, diverticula, and other

small cavity like the posterior urethra it has been found necessary to change the water so frequently that all modern instruments are also designed to be used under a constant irrigation stream. The water enters the instrument through one channel, flows through the urethra, and leaves by another channel. The inlet is connected to an irrigator raised about 18 in. above the level of the patient. Many different instruments are on the market. It is impossible to describe them here, but the author prefers either the Leo Buerger or his own modification of the Geiringer (Fig. 101).

**Technique.**—The instrument should be passed into the bladder, and the irrigation stream turned on. It is best to let this flow for a few seconds before introducing the telescope and switching on the light. With these instruments the base of the bladder as far up as the ureteric orifices can be inspected. As the urethroscope is slowly withdrawn, the neck of the bladder and the posterior urethra are examined.

**Indications.**—(1) The diagnosis and treat-

## URINARY CALCULI

ment of chronic inflammatory conditions by means of topical applications. (2) The destruction of papillomata and polypi, which are common in this locality. This is best effected by means of diathermy. Papillomata just within the sphincter of the bladder, where they are practically out of reach of the ordinary cystoscope, can be fulgurated through a posterior urethroscope.

J. SWIFT JOLY.

**URIC ACID** (*see* URINE, EXAMINATION OF).

**URINARY CALCULI.**—The substances entering into the composition of urinary calculi are uric acid, urates of ammonia and soda, calcium oxalate, calcium phosphate, calcium carbonate, ammonia-magnesium phosphate, cystin, xanthin, indigo. Calculi formed of the last three substances are very rare.

Crystals of uric acid, calcium oxalate, etc., may be passed for years in the urine without any tendency to stone-formation, a colloid substance being necessary to bind them together. *Colloids* are of two varieties, reversible and irreversible; the latter are necessary for the formation of stone, but they are never found in normal urine. The most important of these is *fibrin*, derived from the soluble fibrinogen; it occurs in the urine in inflammatory conditions, or in hæmaturia. It is probable that stone-formation is preceded by inflammation, but once a calculus is formed bacterial inflammation is no longer necessary for its growth, as sufficient fibrin is produced by the mechanical irritation of the walls of the cavity in which it lies.

**Etiology. Geographical distribution.**—Not only does the relative frequency of calculous disease vary in different countries, but in almost every State there are local "stone areas." In the British Isles they are most common in Scotland, the east coast of England, North Wales, and the London district. They are rare in Ireland and the south-west of England.

**Personal.**—Several members of a family may suffer from calculous disease. Vesical calculus is common among children of the poor, but very rare among those of parents in easy circumstances. In adult life it is equally common in all grades of society. Three-quarters of all renal or ureteric calculi are found between the ages of 30 and 60, while vesical calculi occur in childhood, in middle age, and in old men as a complication of hypertrophy of the prostate. Over 95 per

cent. of stones in the bladder are found in men, while renal calculi are almost as common in the female as in the male. Excess in food, a sedentary life, and hard drinking-water are predisposing factors.

*Renal and ureteric calculi* are relatively much more common than was formerly supposed. Out of 713 consecutive cases of stone admitted into St. Peter's Hospital, 217 were either renal or ureteric. Renal calculus is bilateral in about 12 per cent. of cases, and ureteric in about 4 per cent. The vast majority of renal and ureteric stones met with during operation are composed of calcium oxalate, or a mixture of oxalates and phosphates, while most of those passed naturally are formed of uric acid or urates.

**Pathology.**—*Primary* calculi are formed apart from recognizable antecedent changes in the urinary organs, and always originate in the kidneys. *Secondary* calculi are due to inflammation, foreign bodies, etc., and may occur in any part of the urinary tract. Renal calculi probably originate either in the collecting tubules or in a calyx, and remain there for a variable time, but sooner or later tend to be swept down the ureter. Stones smaller than hempseed may be passed painlessly, and are usually termed gravel, but when larger produce renal colic in their passage down the ureter. If a stone remains in the renal pelvis it is at first round or oval and lies freely within it. Such a stone acts as a ball valve, and consequently gives rise to considerable pain. Sooner or later it becomes impacted, the site of impaction being almost always at the junction of the ureter and pelvis. Once fixed it grows rapidly in two directions, upwards into the kidney, and downwards along the ureter, the former being the more important. The stone thus soon becomes triangular in outline as it fills the renal pelvis; later, little projections appear opposite the mouths of the different calyces, which they finally fill and to which they become moulded. At the same time the downward growth produces a beak projecting down the ureter for a variable distance. In this way a complete cast of the renal pelvis and calyces is formed. These stones often cause no obstruction to the urinary flow, but fit snugly into the pelvis, only dilating it by the pressure of their growth. If, however, the calculus becomes so tightly wedged in the orifice of the ureter as to obstruct it, the kidney dilates and a hydro- or a pyonephrosis is formed, according to whether in-

## URINARY CALCULI

fection is absent or present. The flow of urine is then so much diminished that the stone ceases to grow, and thus we have the anomaly of a greatly enlarged kidney rendered useless by a small calculus. In calculous pyonephrosis, secondary calculi often form in the dilated calyces. A certain amount of perinephritis is always produced. If the urine is aseptic, or only slightly infected, this merely results in a slight thickening of the fat around the renal pelvis; but if the urine is more seriously infected, two distinct varieties of perinephritis are met with. The first is a fibro-fatty change: the perinephritic fat is increased in amount, infiltrated with fibrous tissue, and becomes hard, very adherent, and prone to invade the kidney through the renal sinus. In the second variety the fat may be wanting and the kidney is bound down to the neighbouring structures by firm dense fibrous tissue. In about one-third of the cases more than one stone is found. When the kidney is not dilated they tend to become faceted from mutual pressure; when dilatation is present they preserve their round or oval form, but even so there is a tendency for one stone to become impacted in the uretero-pelvic junction and to develop the characteristic ureteric beak.

**Symptomatology.**—(1) **A stone passing down the ureter.** This usually produces renal colic, but if the stone is small or the ureter dilated, as occurs most frequently in patients who have passed a large number of small calculi, there may be no symptoms. *Renal colic* usually begins after exercise or jolting, but may occur at any time and even during sleep. It is characterized by a sudden and very severe attack of pain, usually described as "cutting or stabbing," or occasionally as "grinding or boring." It starts in the back, in the angle between the last rib and the erector spinæ muscle, but soon travels round to the front of the abdomen to a point just below the tip of the tenth rib. Thence it radiates along the course of the ureter, and spreads to the groin, testicle, and upper part of the thigh. It never radiates upwards, or inwards as far as the middle line. The pain is excruciating and causes the patient to roll about in agony, his face drawn and haggard, his pulse small, thready, and rapid, and his skin covered with a cold sweat. His temperature rarely rises above 99° or 100° F., and there is little or no prostration. Nausea and vomiting are always present, and constipation is the rule. The urine is scanty and

always contains blood, though perhaps only in microscopic quantities. Strangury and pain at the tip of the penis are only present when the stone is about to pass into the bladder. Renal colic ends when the stone reaches the bladder, at which time all the symptoms, with the exception of the hæmaturia, terminate abruptly. If, however, the calculus is retained in the ureter the colic is incomplete, and the pain then subsides slowly. The patient feels perfectly well until he gets another attack. Renal colic may last for one or two hours to two or three days, and the intervals between the attacks from a few days to many months.

(2) **Aseptic renal calculus.**—More than half of these patients give a history of complete or incomplete attacks of renal colic, which in many cases are repeated. But the most characteristic pain is of a sharp stabbing nature, brought on by exercise, jolting, or fatigue, and relieved by rest. It remains localized to the angle between the last rib and the erector spinæ muscle, and does not radiate towards the groin or the testicle. It may be so severe as to compel the patient to change his mode of life and give up all forms of exercise. The kidney is nearly always tender on palpation, and there is a rough parallelism between the severity of the spontaneous pain and that provoked by examination. Hæmaturia is present in almost every case, but frequently is only found on microscopical examination of the centrifugalized urine. It is increased by exercise and relieved by rest. Crystals and a few leucocytes are usually found in the urine.

(3) **Infected renal calculus.**—Less than half of these patients give a history of renal colic; it often dates from many years previously, and probably occurred before the kidney became infected. After the onset of infection the pain and hæmaturia are both very much reduced in amount. Acute paroxysms of pain do not occur unless a pyonephrosis is present, and usually nothing more than a dull ache or a constant drag in the side is experienced. The kidney, however, remains tender on palpation. Blood, visible to the naked eye, is rarely found, and often no red blood-cells can be seen with the microscope. The deposit consists of pus, bacteria, and crystals. The most common infecting organism is the staphylococcus, the next the colon bacillus. The patients steadily lose ground and become pale, thin, languid, and weak; the evening temperature is raised, and the typical picture of septic absorption is produced.

## URINARY CALCULI

**Diagnosis.**—Since renal and ureteric calculi may give rise to no symptoms whatever, or to symptoms which are very anomalous, the diagnosis in many cases is only made during a routine X-ray examination of the whole urinary tract (see X-RAYS, DIAGNOSTIC USES OF, PLATE 47, Fig. 6, and PLATE 48, Figs. 1, 2, 3). About 95 per cent. of all renal calculi can thus be demonstrated. Uric-acid stones alone are transparent to X-rays, but rarely grow large enough in the kidney to necessitate removal by operation and are almost invariably passed naturally. All cases of pyuria, of hæmaturia, and of definite renal pain should be submitted to an X-ray examination.

Renal calculus must be distinguished (1) from an affection of some other organ and (2) from some other renal condition. Renal colic is most often confused with *appendicitis* or *biliary colic*. The pain in *appendicitis* is at a lower level than that of renal colic, is not felt at the back, and does not radiate down to the testicles. Localized tenderness over McBurney's point and rigidity of the abdominal muscles are useful differential points. The tenderness during renal colic is only elicited by deep palpation in the loin and is felt more in the back, while the muscular rigidity in this condition is slight and confined to the oblique muscles of the abdomen. In both *appendicitis* and *biliary colic* the right rectus is held rigid. Hæmaturia is always present in renal colic, though the blood is often microscopic in quantity, but it is very rare in *appendicitis*. The pain of *biliary colic* is felt under the costal margin at the outer border of the rectus muscle. It radiates to the epigastrium and upwards towards the right shoulder, but never downwards. Rigidity of the abdominal muscles in the right upper quadrant of the abdomen is pronounced, and the upper part of the right rectus is generally like a board, and renders palpation of the gall-bladder very difficult. Renal colic almost indistinguishable from that produced by stone may be caused by the passage of hydatid cysts, bloodclots, masses of caseous material, etc., down the ureter. The most common causes of severe hæmorrhage into the renal pelvis are *trauma* and *renal neoplasm*. In the former case there is a history of an accident; in the latter the bleeding is usually severe but very intermittent, and a hard irregular tumour can generally be felt in the renal region. The painful crises of *hydronephrosis* and *movable kidney* are, as a rule, unaccompanied by bleeding, and the pain tends to remain localized to

the loin. The history of polyuria alternating with oliguria in the former condition, and the abnormal mobility of the kidney in the latter, are important diagnostic points; but in all cases the presence of a stone should be sought for by an X-ray examination.

**Treatment.** (1) **Operative.**—Every stone that gives an X-ray shadow larger than a six-penny-piece ought to be removed without delay, the indications being (a) to relieve the patient's sufferings and (b) to save the kidney from destruction. If the shadow is smaller than this arbitrary limit, operation is still indicated if the pain is excessive or if there is evidence of damage to the kidney, and especially if the calculus is infected. Otherwise the calculus may be left for a time in the hope that it may be passed naturally. If, however, after one or, at the most, two attacks of renal colic there is no evidence of the stone passing down the ureter, operation should no longer be postponed. In all cases every effort should be made to save the kidney, and either a *nephrolithotomy* or a *pyelolithotomy* performed; a primary *nephrectomy* is only indicated when the organ is absolutely destroyed.

(2) **Medical and dietetic measures** are required (a) to prevent a recurrence after operation, and (b) in cases where small stones and gravel are passed naturally. The object is first to diminish the amount of stone-forming salts in the urine, and secondly to modify the urine so that it may best keep them in solution. In the case of uric-acid calculi the first object is gained by diminishing the amount of purin-bearing bodies in the diet. Cellular organs, such as liver, kidneys, sweetbreads, etc., should be absolutely forbidden, and ordinary butcher's meat partaken of only sparingly. The diet should be largely vegetarian, but should not be too strict, or the general health may suffer. All gastro-intestinal irregularities should be carefully corrected. This is most important in the case of oxaluria, as hyperchlorhydria undoubtedly increases the absorption of the vegetable oxalates. The drugs in most common use are the alkalis, lithium salts, and organic substances such as piperazine (diethylene diamine) and hexamine (hexamethylene-tetramine). Although these substances form soluble urates *in vitro*, it is probable that their action is much less effective in the kidneys. In the case of oxalate and phosphatic calculi the chief point in the medicinal treatment is to limit as far as possible the intake of calcium, since this metal alone forms

## URINARY CALCULI

the insoluble salts. All drinking-water should therefore be boiled or, better still, distilled. In all cases of gravel, no matter what its nature, large quantities of bland fluid should be administered. These act (i) by diluting the urine and rendering supersaturation of the urinary salts impossible or unlikely, and (ii) mechanically by flushing out the renal pelvis and washing away any small concretions that may form before they grow large enough to give rise to symptoms. In this respect the mineral waters of Vittel and Contrexéville are of great value. Diuretic treatment is contraindicated, however, in cases in which (a) the calculus is causing hydronephrosis, (b) there is a marked interstitial nephritis, or (c) the bladder is unable to empty itself either on account of paralysis, as in locomotor ataxy, or because its outflow is obstructed, as in cases of enlarged prostate or stricture of the urethra.

(3) **Treatment of renal colic.**—The objects are to relieve the pain, to diminish the spasm of the ureter, and to favour the expulsion of the stone in cases in which it is small enough to pass naturally. Morphia is usually administered, but must be given in large doses before the desired effect is obtained. It has recently been shown that some of the other alkaloids of opium are much more efficacious than morphia in relieving the spasm of the ureter. Papaverine is the most effective, but it is not necessary to administer this alkaloid in its pure state, as the same clinical effect is obtained by the use of opium or omnipon. Hot baths or hot compresses are of great value. Diuretics should not be given unless it is known that the stone is small enough to be passed naturally. Catheterization of the ureter has in a few cases put a sudden stop to the pain of a renal colic. This only happens if the catheter can be passed above the stone, or if it displaces the latter upwards either into the renal pelvis or into a dilated portion of the ureter. In either case renal tension is relieved and the pain suddenly stops. However, the stone is generally held so tightly by the spasm of the ureter that it cannot be dislodged.

**Prognosis.** (1) *Operative mortality.*—There have been 8 deaths following operation in 217 consecutive cases of renal or ureteric calculi admitted to St. Peter's Hospital, and all of these occurred in cases where the kidney was badly infected. In no case did death follow a simple pyelotomy. Thus the removal of an

uninfected stone may be considered as devoid of risk. (2) *Recurrence.*—I have no accurate statistics on this point, but recurrence nearly always takes place when the kidney is infected before, and remains infected after, the operation. In aseptic cases recurrence is very rare. Recurrent stones are nearly always phosphatic. (3) *Cases where no operation is performed.*—In these cases the expectation of life is always lessened. Renal calculus has a distinct tendency to become bilateral in the late stages, and patients are liable to succumb to anuria, septic pyelonephritis, septic intoxication, or nephritis.

Urinary calculi having been considered generally, the special features of Calculous Anuria, Ureteric Calculus, Vesical Calculus, Prostatic Calculus, Stone in the Urethra, and Preputial Calculus must now be described.

### CALCULOUS ANURIA

This serious condition is, I believe, becoming less common since renal calculi have been recognized earlier, and especially since the introduction of the X-rays. The patients usually give a history of renal colic or a lumbar ache, but in the majority of cases they have been free from pain for a considerable time before the attack. The anuria comes on suddenly; the patient notices that he has not passed water for several hours, and then finds that he can void only a few teaspoonfuls. In many cases he has followed his vocation for two or three days before seeking advice. The anuria is rarely complete, a few drachms of urine being usually passed every day. For the first five or six days there is a period of tolerance in which the patient feels in his usual state of health. Then he notices a loss of appetite, a feeling of lassitude, and insomnia. The period of intoxication follows, and the symptoms of uræmia appear—headache, insomnia, great muscular weakness and prostration, complete loss of appetite, dry mouth and tongue, and muscular twitching. Soon the patient suffers from drowsiness, which deepens into coma. Delirium and convulsions sometimes occur, but as a rule he becomes deeply comatose, and dies on about the tenth or twelfth day from the onset. Calculous anuria is caused by a stone blocking one kidney while the other has either been destroyed by disease or removed by operation, or else by calculous disease of both kidneys. It is distinguished from simple retention by means of the catheter, and from

## URINARY CALCULI

anuria due to pressure on the ureters by a tumour, such as a cancer of the uterus, by physical examination. The most difficult point is to ascertain which kidney was the last to function. This is determined partly by the history of the case. The kidney in which pain was last felt is usually the one blocked, as a functionless kidney is generally painless. Again, rigidity of the abdominal wall is of great help, as it is found in all cases where a kidney has recently been blocked. An X-ray examination should be made immediately, for it will show not only which side was the last to be blocked, but also where the stone is. Usually it is found in the upper part of the ureter. If an X-ray examination cannot be made immediately, the kidney in which pain was last felt should be exposed and drained. This will permit a more thorough examination. The mortality from calculous anuria in cases not operated on is over 70 per cent.; if operation is performed within thirty-six hours it is about 10 per cent., and it rises approximately 5 per cent. for every day the operation is delayed.

### URETERIC CALCULUS

The *rapid* passage of a stone down the ureter has been discussed in connexion with renal colic. If it passes *slowly* down the ureter there are several attacks of colic, and the patient soon notices that the site of the pain changes. At first it is felt in the loin, whence it radiates downwards; later on the pain commences in front, close to the outer border of the rectus. If the stone is in the lumbar portion of the ureter the pain is felt at the level of the umbilicus, and if it lies in the extreme lower end the pain may be just over the external abdominal ring. As the stone passes downwards the pain may be felt anywhere between these points. If it is impacted the pain may be fixed instead of radiating to the testicle; in this case there is usually no renal pain unless the kidney is either distended or infected. A stone in the intramural portion of the ureter usually gives rise to vesical symptoms—frequency of micturition and pain during the act—in addition to those described above. Changes in the urine—i.e. the presence of blood or pus, or both—are found in over 80 per cent. of cases, and cystoscopic changes are noticed in nearly 90 per cent. The diagnosis is completed by means of the X-rays (see X-RAYS, DIAGNOSTIC USES OF, pp. 561-2), but it must be remembered that shadows like those thrown by stones in the

pelvic portion of the ureter may be caused by phleboliths or calcareous glands. In doubtful cases the patient should be re-examined with an opaque bougie in the ureter.

The ideal **treatment** consists in removing the stone before the corresponding kidney becomes seriously damaged, and, as this is liable to happen earlier in cases of ureteric than of renal calculi, operation should not be postponed too long. All cases of large ureteric calculi in which the shadow is larger than a sixpenny-piece, and all cases in which the stones are causing either infection or dilatation of the kidney, should be operated upon without delay. If there are repeated attacks of colic the patient should be X-rayed after each attack, and, if the position of the stone does not change, operation is indicated. An attempt should be made to encourage small ureteric calculi to pass naturally; diuretics and regular exercise should be prescribed; at the same time an attempt should be made to mobilize the stone by means of a ureteric catheter, and its descent should be facilitated by injection either of lubricants or of local anaesthetics up the ureter. The mortality after the removal of a stone from the ureter is very small. In the Mayo clinic it is  $\frac{1}{2}$  per cent. (2 deaths out of 400 cases), and at St. Peter's Hospital, London, it has been nil during the last ten years.

### VESICAL CALCULUS

A stone in the bladder may (a) derive from a renal calculus that has passed down the ureter, in which case it simply grows in the bladder, or may (b) originate in the bladder itself, when it is due to infection or to a foreign body. The latter are almost invariably phosphatic, while the former are composed of uric acid, oxalates, or (occasionally) phosphates. Uric acid stones are usually single, ovoid in shape, and dark reddish-brown in colour, and on section show a laminated structure. They are smooth, very slightly nodular, heavy, and hard. Urate stones are similar, but lighter in colour. Oxalate calculi are also usually single, but very hard and heavy; they are round and covered with rough nodules or long sharp spines, while their colour varies from brown to black and is due to blood pigment; they are often termed "mulberry calculi." Phosphatic stones are white or light grey in colour, chalky in appearance, and soft and friable. If more than one substance enters into the composition of a stone it is called a mixed calculus; for ex-

## URINARY CALCULI

ample, a stone may have a nucleus of calcium oxalate which is covered by a thick layer of uric acid, and this in turn may be covered by a layer of phosphates. After the onset of cystitis all stones tend to be covered with a layer of phosphates, so that mere inspection gives very little idea of their composition.

Most vesical calculi lie free in the cavity of the bladder, and when examined with the cystoscope are found to be resting on the bladder-wall at a point immediately behind the trigone. A stone may lie in a diverticulum and grow there until it completely fills it, subsequent growth being outwards into the bladder. In this way dumb-bell-shaped stones are formed, the middle of the dumb-bell being shaped by the constricting neck of the diverticulum.

**Symptomatology.** (1) There is *frequency of micturition*, increased by exercise or jolting and relieved by rest. It is therefore more pronounced by day than by night. This is the reverse of the frequency due to enlarged prostate, which is worst by night. It is also distinguishable from the frequency of tubercle or malignant disease of the bladder, as in those diseases the frequency is both diurnal and nocturnal.

(2) *Pain* is intimately connected with the act of micturition, is experienced just as the bladder is emptied, and is localized to a point near the frænum of the penis, on the under-surface about half an inch from the tip. It commences when the urine is passing, reaches a maximum when the bladder is emptied, and dies away in about ten or fifteen minutes. It is described as "sharp or cutting." In addition a dull aching pain is often felt in the bladder itself, and is increased by exercise or jolting. The pain due to stone in the bladder is most acute in children, not so severe in middle age, and often hardly noticeable in old men who are suffering from enlarged prostate.

(3) *Hæmaturia* is rarely profuse, but is noticed at almost every act of micturition. It is a "terminal" hæmaturia; when the bladder is almost empty the blood appears in the urine, and the last few drops evacuated are almost pure blood. The amount of blood passed is increased by exercise or jolting.

(4) *Interruption of the jet* is an important symptom in children and women, in whom the stone comes into contact with the internal meatus, but it is not so prominent in middle-aged or old men, as the prostate prevents the calculus from impinging on the opening of the

urethra. It is not due to an actual blocking of the urethra by the stone, but to a sudden spasm of the sphincter from the irritation of contact between the stone and the sensitive mucous membrane at the orifice of the urethra. When this symptom is pronounced there is a sudden and painful stoppage of the jet, which only occurs when the urine is flowing freely. In a few seconds the stream recommences, at first slowly, but soon recovering its former volume. It is rare for the stream to be interrupted twice in one act of micturition. This sudden stoppage of the jet must be distinguished from the type of interrupted micturition often observed in cases of stricture or enlarged prostate. In these disorders the stream, which is always poor, slowly dies away to a dribble and then gradually ceases altogether, and is only restarted by a violent contraction of the detrusor. It is an indication that the muscle is almost unable to overcome the obstruction and soon tires in its task.

In every case of vesical calculus that is left to itself, cystitis sooner or later supervenes. The urine becomes purulent and deposits a thick sediment of mucus on standing. In certain cases the coating of the stone with phosphates somewhat relieves the symptoms, as, for example, when a rough oxalate stone is covered by a comparatively smooth layer of phosphates. After the advent of cystitis the frequency of micturition is nocturnal as well as diurnal, and as the bladder becomes more and more irritable the frequency increases until it ends in incontinence.

About 7 per cent. of the patients with enlarged prostate who come to operation have a stone in the bladder. In such cases the symptoms of stone are profoundly modified. If the patient has residual urine the characteristic pain at the end of micturition is wanting, as in this case the bladder is never completely emptied, and the only symptoms are hæmaturia after walking or jolting, and a deep-seated pain either in the bladder or the perineum. If he is on catheter life, in addition to the symptoms just mentioned he feels pain at the tip of the penis every time he empties his bladder.

**Diagnosis.** - For *sounding*, the bladder should contain two or three ounces of fluid. The sound is introduced and usually grates almost immediately against the stone. If it is not felt immediately the bladder should methodically be searched. Sounding reveals the majority of calculi, but is not so accurate



## URINARY CALCULI

or exact in the information it gives as is the *cystoscope*. This instrument will tell the number, size, position, and in many cases the composition of the stones. It also determines whether a calculus is movable or fixed. If these methods fail, an *X-ray examination* should be made; this is especially useful in children. In the child the bladder can be palpated between a finger in the rectum and a hand on the abdominal wall.

**Treatment.**—An uncomplicated stone up to  $1\frac{1}{2}$  in. in diameter can be crushed; this operation (*litholapaxy*) is of immense advantage to the patient, as the convalescence after it is short (four or five days). If the stone is too large to be grasped by the lithotrite, if the urethra is too narrow to admit the instrument, if the stone is fixed, or if some other condition such as an enlarged prostate is present and necessitates opening the bladder, a *suprapubic lithotomy* should be performed. The old perineal operations for stone have now been abandoned.

**Prognosis.**—The mortality of all cases of stone in the bladder admitted into St. Peter's Hospital is now a little less than  $2\frac{1}{2}$  per cent. The prognosis as to recurrence depends largely on the state of the bladder. If the cystitis is relieved and the bladder able to empty itself satisfactorily, the risk of recurrence is small. If, however, the prostate is enlarged, the patient is very liable to recurrence unless the gland is removed and the obstruction relieved.

### PROSTATIC CALCULUS

True prostatic calculi, calcified corpora amylacea, are very common, but as they give rise to no symptoms they need no consideration.

A urinary calculus may lodge in the prostatic urethra, or in a cavity in the prostate resulting from an old gonorrhoeal or tuberculous abscess. If left alone it tends to grow upwards along the urethra, and finally to project into the bladder. This growth is due to the deposition of urinary salts.

**Symptoms.**—(1) Pain on micturition resembling that of vesical calculus. (2) Pain in the perineum, especially when sitting. (3) Alteration in the act of micturition. Patients sometimes can only pass water in the sitting position, or may find it necessary to press deeply in the perineum so as to dislodge the stone. Interruption of micturition is sometimes noticed. If the stone dilates the sphincter, incontinence may be present.

**Diagnosis.**—The stone is felt either by

## URINE, EXAMINATION OF

means of a sound in the urethra or by the finger in the rectum.

**Treatment.**—If the stone is not fixed it should be pushed back into the bladder by means of a large steel sound, and then crushed. If it is fixed either in the prostatic urethra or in the cavity in the prostate, it should be removed by a perineal section.

### STONE IN THE URETHRA

A stone may become impacted in the anterior urethra either behind a stricture or in the fossa navicularis. In either case it can be felt by palpation, or by means of an instrument in the urethra. If it is lodged behind a stricture an external urethrotomy should be performed; if it lies in the fossa navicularis it is best to do meatotomy and remove it by means of fine forceps.

### PREPUTIAL CALCULI

These are either composed of smegma impregnated with lime salts, or are true urinary calculi caught under a tight foreskin. Phimosis is always present, and a profuse purulent discharge comes from under the prepuce. The stones are diagnosed by palpation. The treatment is circumcision (*see* PHIMOSIS).

J. SWIFT JOLY.

### URINARY DEPOSITS (*see* URINE, EXAMINATION OF).

### URINARY FISTULÆ (*see* VAGINAL AND UTERINE FISTULÆ; URETHRAL STRICTURE).

### URINARY SCHISTOSOMIASIS (*see* SCHISTOSOMIASIS).

**URINE, EXAMINATION OF.**—The factors needed in reading an analysis of the urine are the amount passed in twenty-four hours, the quantity of each constituent contained in this amount, and the relations existing between them. The influence of the diet, the water ingested, the temperature, the physical state of the patient, the age, the sex, and the amount of exercise must be borne in mind also in interpreting the results. Sometimes it is advisable to examine in addition a specimen of the urine collected at some particular time when it is likely that intermittent traces of an abnormal constituent will be present. Thus sugar may be demonstrated in a specimen taken three or four hours after a carbohydrate meal when a twenty-four hours'

## URINE, EXAMINATION OF

sample would give a doubtful or negative result, and by comparing the urine voided on rising with that passed at the end of a day's work the existence of slight chronic nephritis and cyclic albuminuria may be shown.

The **specific gravity** is determined by the urinometer, which gives the weight of the urine relatively to an equal bulk of water, which is usually expressed as 1,000. It is important that the urine should be at the standard temperature of 15° C., or that allowance should be made for any deviation from that standard. The specific gravity of a mixed twenty-four hours' specimen of normal urine varies between 1,015 and 1,025. In the new-born child it is much lower, 1,005 to 1,007. As a rule, only the result obtained with a twenty-four hours' specimen is of much value, since different portions taken during the day and night may vary from 1,002 to 1,040. Occasionally an examination of a separate specimen may be helpful. In the diagnosis of early chronic diffuse nephritis, for instance, the constant low specific gravity of the morning urine is of value. Generally the specific gravity varies inversely with the amount of urine passed, but not strictly so, as an increased output of fluid always raises the excretion of solids to some extent. In diabetes mellitus, however, there usually is an increase in the specific gravity, 1,025 to 1,040, with an increase in the quantity of urine, although it is important to remember that sugar may be present with a specific gravity as low as 1,006 or even 1,002. In nephritis with renal insufficiency, on the other hand, a diminution in the specific gravity with scanty urine may occur and suggest impending uræmia. The specific gravity is always very low in diabetes insipidus and the amount of urine excessive. A constant low specific gravity is a sign of a number of conditions in which the kidney tissue is greatly diminished.

A high specific gravity generally indicates an excess of urea, sodium chloride, or the presence of sugar.

The **colour** of the urine is influenced by its dilution and therefore varies with the specific gravity, being pale in dilute and high-coloured in dense urines. A noteworthy exception is diabetes mellitus, in which the passage of a large amount of pale urine with a high specific gravity is suggestive. Acid urines are usually more high-coloured than alkaline urines of the same density. As a rule, pale urine excludes the existence of a febrile disease of any severity, while the continued excretion of

very pale urine is usually associated with more or less anæmia.

When there is rapid destruction of the erythrocytes, the urine is high-coloured from the presence of an excess of urobilin. This pigment is also responsible for the heightened colour of the urine passed after blood extravasations, blood poisoning, in febrile conditions, chronic passive congestions, lead poisoning, cholangitis, cirrhosis, and other liver conditions. Distinctly *red* tints of the urine are always due to the presence of foreign colouring matter, most often hæmoglobin. If traces of methæmoglobin are also present, the urine has a characteristic smoky appearance. The ingestion of eosin colours the urine red. Drugs containing chrysophanic acid also give a red colour if the urine is alkaline, but yellow if it is acid. *Dark-brown* urines may arise from the presence of urobilin, larger quantities of methæmoglobin, or the ethereal sulphates such as that of indoxyl. They do not blacken with ferric chloride nor readily reduce copper solutions. The *blue* colour sometimes seen in cases of this description is due to a higher oxidation product of indoxyl.

*Black* urines are met with in longstanding jaundice, hæmaturia, and hæmoglobinuria, melanotic sarcoma, alkaptonuria, ochronosis, from great excess of indoxyl sulphate, and after the use of phenol and phenol derivatives; but only those met with in melanotic sarcoma and alkaptonuria are truly black. In many instances the urine is of normal colour when voided, the darkening taking place on standing. With melanin the change is hastened by the addition of nitric acid, with indoxyl-sulphate by warming with nitric acid, and with alkaptonuria by the addition of an alkali. Urines containing melanin give with ferric chloride an immediate blackening and a grey precipitate soluble in excess, whilst alkapton urines show an evanescent deep blue coloration and darken with the addition of each drop of the reagent. Urines containing pyrocatechin and hydrochinon, also alkapton urines, reduce solutions of copper, but not bismuth.

*Yellow, brown, green, and greenish-black* urines are passed in jaundice, the colour depending upon the extent of the obstruction and degree of oxidation of the pigments present. The urine is often of a light greenish hue in diabetes, especially if there be a high percentage of sugar. The urine first passed after the administration of methylene-blue is green, later it becomes blue. A *dull-blue* tint is not

## URINE, EXAMINATION OF

uncommon in cholera and typhus. The *salmon-pink* colour of the urates deposited after excessive exercise, a rich meat diet, sweating, etc., is due to uroerythrin. *Milky* urine is passed in chyluria, and may also arise from the presence of phosphates or pus, but the latter deposits on standing.

The **reaction** of the urine is taken with litmus paper for qualitative work, while phenolphthalein is employed as the indicator in quantitative examination. A well-preserved twenty-four hours' specimen of normal urine is faintly acid to litmus and requires from 30 to 40 c.c. of decinormal sodium hydrate solution to neutralize each hundred cubic centimetres, with a total of 300 to 600 c.c. using phenolphthalein as the indicator. The acidity of the urine, as it is commonly understood, depends for the most part upon the presence of acid salts, particularly diacid sodium phosphate, and not upon the presence of free acids. The reaction is influenced considerably by the diet, the acidity being greater the more protein is oxidized, and less the larger the quantity of alkali-forming foods taken. It is highest in the morning before breakfast and lower a few hours after each meal. The urine passed two to four hours after a meal may be alkaline ("the alkaline tide") and show a turbidity from the presence of phosphates of the alkaline earths. It is never neutral in reaction, but occasionally a specimen turns red litmus blue and blue litmus red ("amphoteric"). This is due to the simultaneous presence of diacid sodium phosphate (acid) and disodium phosphate (alkaline). Physiologically, the acidity of the urine is increased by abstinence from fluids, by a nitrogenous diet, from circumstances increasing the concentration, and after the ingestion of mineral, benzoic, or boric acids. A pathological increase in the acidity may be found in diabetes mellitus, in diseases which increase tissue waste, in certain hepatic diseases, in scurvy, leukaemia, gout, rheumatism, and chronic nephritis. Persistent hyperacidity is a frequent sign of renal lithiasis. The acidity is diminished physiologically by copious draughts of liquid, by a non-nitrogenous diet, and following the ingestion of vegetable acids and salts excreted as alkaline carbonates. Pathological diminution of the acidity is found in diseases of the stomach, after vomiting, in diabetes insipidus and other polyurias, when transudates are being absorbed, in neurasthenia, anemia, some abdominal disorders, in severe chronic parenchy-

matous nephritis with oedema, and after hæmorrhage into the intestine. It is also alkaline when mixed with alkaline secretions from the urogenital tract. Lastly, it may undergo alkaline decomposition within the bladder or after it has been passed, the alkalinity being due to the presence of ammonia formed by the breaking down of urea by bacteria. To determine whether the alkalinity of a specimen is due to fixed alkalis or ammonia, the litmus paper is dried; if the red colour return, ammonia is present; if it remain blue, a fixed alkali is the cause.

The **total mineral ash** of the urine varies from 9 to 25 grm. per day. **Chlorides**, next to nitrogen, are the principal constituent of the urine, forming nearly one-quarter by weight of the total solids. Sodium chloride is the chief chloride. The amount of chlorides excreted depends largely on the quantity ingested, but, since the osmotic pressure of the tissue fluids is maintained at a constant level mainly by variations in the amount of sodium chloride excreted, anything tending to disturb the osmotic equilibrium of the body is liable to alter the excretion of chlorides in the urine. The chlorides excreted in 24 hours vary from 10 to 15 grm., but persons fond of salt may pass 40 to 50 grm. Retention of chlorides occurs in fevers, especially toward the crisis. A great diminution or absence suggests pneumonia. A low figure, 2 grm. or less in the day, is a bad sign in any chronic disease. In gastric disorders, an excess of urea relative to chlorides (2 or more to 1) suggests cancer. In acute rheumatism, the disappearance of chlorides, without increased joint-involvement, suggests pericarditis with effusion.

**Estimation.**—Chlorides may be roughly estimated by acidifying albumin-free urine with nitric acid and adding one drop of 10-per-cent silver nitrate. If the chlorides are normal or increased the precipitate sinks as a compact ball, but if they are diminished it separates. Quantitative determinations are best carried out by Arnold's modification of Volhard's method, but Purdy's centrifuge method gives results by which the changes from day to day may be compared.

**Phosphates** are present in the urine as salts of the alkaline bases and as earthy phosphates. The total amount varies from 1 to 5 grm. in the twenty-four hours. The earthy phosphates vary from 1.0 to 1.5 grm., the alkaline from 2.0 to 4.0 grm. The ratio of urea to phosphoric acid ranges from 8-12 to 1, the ratio

## URINE, EXAMINATION OF

of phosphoric acid to nitrogen from 17–20 to 100. The constitution and state of the phosphates vary with the reaction of the urine. In acid urines all the phosphates are in solution as “acid” phosphates, some 60 per cent. occurring as dihydrogen phosphate and some 40 per cent. as the monohydrogen salt. When the proportion of the former is reduced to 30 or 40 per cent., the urine becomes alkaline or amphoteric. In alkaline urines, normal phosphates containing no hydrogen appear. Such urines may be turbid with a white sediment of earthy phosphates, a condition known as “phosphaturia” or, better, “alkalinuria,” since the total phosphoric acid content of the urine is not necessarily raised and the precipitation is due to the diminished acidity. It is met with in persons on a vegetable diet, where there is loss of hydrochloric acid, and as a symptom of neurasthenia. The earthy phosphates are precipitated from feebly acid, amphoteric, or alkaline urines by boiling, and from all urines by caustic alkalis. Some 50 mg. of phosphorus are excreted daily in an organic form (glycero-phosphoric acid).

The phosphates of the urine are derived partly from the inorganic phosphates of the food, and partly from metabolism of phosphorus-containing materials of the food and tissues. A proportion of the phosphates ingested, varying from 12 to 50 per cent., is excreted in the feces. The higher the calcium and magnesium content of the food, the lower the absorption and the excretion of phosphates in the urine. They are therefore diminished by a milk or vegetable diet and increased by a meat diet. The reaction of the body fluids is maintained partly by variations in the amount of phosphates excreted in the urine, and an increase is always seen in conditions tending to produce acidosis.

“*Phosphatic diabetes*” is the term applied to a condition in which 3.5 to 4.0 gm. at least of phosphoric acid are passed daily, without there being any sugar. Some of the cases are cases of glycosuria in which the sugar is temporarily absent, others are cases of nervous polyuria. The total phosphates are often decreased much more than the urea in nephritis. In Addison's disease the ratio of urea to phosphoric acid is often greatly increased.

**Estimation.**—An approximate determination of the phosphoric acid may be made by adding ammonia to a test-tube half-full of filtered urine, warming, and standing overnight. A precipitate  $\frac{1}{4}$ – $\frac{1}{2}$  in. deep of earthy phosphates

is normal. This is then filtered off, one finger's breadth of magnesia mixture is added to the filtrate and the mixture warmed. If, after 18–24 hours, the precipitate measures  $\frac{1}{2}$ – $\frac{3}{4}$  in. deep, the alkaline phosphates are normal. Figures for comparative purposes can be obtained by Purdy's centrifugal method, but for accurate determinations the phosphates must be titrated with a standard solution of uranium nitrate.

**Sulphates** are derived chiefly from the oxidation of sulphur-containing substances, mainly proteins; only a small portion comes from sulphates taken in the food. The sulphate content of the urine is, therefore, a rough measure of the total protein metabolism. The ratio of nitrogen to sulphuric acid is usually about 5 to 1.

Sulphates occur in two forms, (a) *preformed*, *mineral*, or *neutral*, and (b) *etheral*, *aromatic*, or *conjugated*. The former are compounds of sulphuric acid with alkalis, the latter are esters formed by the union of sulphuric acid with aromatic substances. The proportion of *etheral sulphates* varies, but in a normal person on a mixed diet they average about one-tenth of the total, which ranges from 1.5 to 3.0 gm. in the twenty-four hours. During starvation, and on diets relatively poor in protein, the proportion increases. A marked increase follows the administration of certain phenolic substances, and also occurs when such compounds are formed in the body by the action of bacteria, as in intestinal obstruction, chronic intestinal catarrhs, and some cases of constipation, and pus absorption. They are diminished on a milk diet, in acute catarrh of the intestine and by purgatives such as calomel. In gastric diseases, even when there is much stagnation and fermentation, the etheral sulphates are little affected. The *preformed sulphates* are increased by a meat diet, by active exercise and oxygen inhalations, in acute febrile and inflammatory diseases, and in diabetes mellitus and insipidus. They are diminished by fasting, a vegetable diet, the use of phenol-containing substances, in convalescence from acute diseases and in chronic diseases. *Neutral* or “*unoxidized*” sulphur occurs to the extent of about 50 mg. daily. The quantity is independent of the diet. Starvation and the breakdown of tissue proteins by toxic substances cause a relative increase. In obstructive jaundice the neutral sulphur may constitute 25 to 60 per cent. of the total. A similar increase is met with in cystinuria, a condition

## URINE, EXAMINATION OF

due to an inborn error of metabolism which results in imperfect breaking down of some of the earlier protein fractions and their excretion as cystin.

**Estimations** of the sulphur and sulphates are not easy to carry out, and although a disturbance of the normal relation between the ethereal and total sulphates is suggestive of abnormal decomposition in the intestinal tract, similar information can be secured by less laborious methods. Putrefaction in the intestinal contents gives rise to indol; this on entering the blood is oxidized to indoxyl, which combines with sulphuric acid and appears in the urine as indoxyl-potassium-sulphonate, or indican. Skatol, or skatoxyl, arises in a similar way. These substances differ from other ethereal sulphates, which are normal tissue metabolites, in being bacterial-decomposition products of tryptophane, so that the quantity excreted affords a measure of the amount of putrefaction in the intestine, or elsewhere.

**Indican** and **skatol** are readily tested for, and the quantity present may be estimated by comparing the depth of colour obtained with a series of standard dilutions or with a standard solution in a colorimeter. Of the numerous tests for **indican** I have found the following the best. To 5 c.c. of the urine add, in this order, one drop of 0.5 per cent. potassium chlorate solution, 2.5 c.c. of chloroform, and 5 c.c. of *fuming* hydrochloric acid (sp. gr. 1.19), mix well by pouring from one tube to another, but do not shake, observe the maximum blue colour in about ten minutes. For **skatol**, mix the urine with an equal volume of a solution containing dimethyl-amidobenzaldehyde 0.33 gm., fuming hydrochloric acid 50 c.c., distilled water 50 c.c., boil, cool, and make alkaline with ammonia. The intensity of the red colour varies with the skatol content of the urine. Normally the urine contains 5 to 15 mg. of indican and 5 to 10 mg. of skatol in the twenty-four hours when a mixed diet is taken. As a rule skatol is more abundant when the large intestine is involved, indican when the upper small intestine is affected. Both are then lessened by a milk diet and by bismuth. When indicanuria is due to foci of pus, diet has no effect, and the largest quantity is found at night.

The **oxalic acid** in normal urine varies from 20 to 35 mg. in the twenty-four hours. It is increased by the ingestion of certain fruits and vegetables and, pathologically, in digestive disturbances with excessive fermentation of

carbohydrates, in icterus and various hepatic and respiratory diseases. In diabetes mellitus the oxalic-acid excretion may rise to 1.5 gm. a day and be associated with a diminution or disappearance of the sugar ("vicarious oxaluria"). The oxalic acid of the urine is combined with calcium and is held in solution by diacid phosphate of sodium, but at times it may appear in the sediment. A copious oxalate sediment does not necessarily, however, signify an increase in the oxalic acid. In the condition known as *oxaluria* numerous crystals are found, sometimes with traces of albumin. It is apparently due to defective carbohydrate digestion with a deficiency of hydrochloric acid, the albumin resulting from irritation of the kidneys.

When carbohydrate metabolism breaks down or the glycogen store of the liver is depleted, a series of intermediate products in the combustion of fatty acids, known as the **acetone bodies**, appears in the urine. **Acetone**, the first of these, is a normal constituent, occurring to the extent of 0.01 to 0.03 gm. in the twenty-four hours. It is increased in hunger, when the carbohydrates of the food are limited, in some fevers, after chloroform and ether anaesthesia, particularly in children, and in other conditions, but especially in persistent glycosuria.

**Tests.**—Many tests for acetone have been described, but the most satisfactory is *Rothera's modification of Legal's*. Half a test-tubeful of the urine is saturated with solid ammonium sulphate, a few drops of a fresh solution of sodium nitro-prusside and a drop or two of acetic acid are added. Strong ammonia is then run on to the surface. In the presence of acetone a purple ring develops at the junction.

**Estimation.**—The quantitative determination is most easily carried out by the Folin and Denis process, in which the density of the precipitate obtained on adding Scott-Wilson's reagent to the acetone aspirated from a known volume of the urine into a solution of sodium bisulphite is compared with that given by a standard solution of acetone.

**Aceto-acetic or diacetic acid** appears and increases in amount as the metabolism of fatty acids becomes more defective. Its presence is always pathological. As a rule, the prognosis in diabetes mellitus is grave when aceto-acetic acid is persistently present, especially in young people and in emaciated patients. Traces may be met with when regulation of the diet is commenced, and, later, may disappear if the intake of carbohydrate and fat is carefully

## URINE, EXAMINATION OF

controlled. The aceto-acetic acid occurring in the urine of children and young people in the course of fevers and digestive disorders is often of little importance, but a similar condition in adults is of grave significance. The treatment of gastric ulcer by starvation and rectal feeding is sometimes followed by the appearance of aceto-acetic acid, but it quickly disappears when carbohydrates are given by the mouth.

**Test.**—*Gerhardt's test* is the one usually employed for aceto-acetic acid, a Bordeaux-red colour being given with a solution of ferric chloride. Fresh acid urine should be used, and the phosphates precipitated by the first few drops of the solution should be filtered off. A number of substances, including salicylates, aspirin, antipyrin, etc., give a similar reaction. A comparison of the depth of colour obtained with the fresh urine and that given by a specimen which has been kept in a tightly corked bottle overnight will often help to show to which a positive reaction is due, since aceto-acetic acid is partly converted into acetone on standing, and so gives a less marked reaction.

**Estimation.**—The simplest process for estimating aceto-acetic acid is Folin and Denis' "turbidity" method. The aceto-acetic acid is converted into acetone by heating it with dilute sulphuric acid and the total acetone then determined.

When the acetone in the urine reaches 0.4–0.5 gm. a day, aceto-acetic acid is always present. When it rises to 0.6 to 1.0 gm., a third substance, **beta-oxybutyric acid**, is usually met with. In severe cases of diabetes 50 to 100 gm., and rarely 200 gm., may be passed in 24 hours. It is also found in scurvy, scarlet fever, measles, insanity, and starvation.

**Test.**—No simple test for oxybutyric acid exists, but since it is laevorotatory, its presence may be suspected when polariscopic determination of the sugar in the urine gives a lower result than titration.

**Estimation.**—Folin and Denis' modification of Shaffer's oxidation method by potassium bichromate and sulphuric acid, with a turbidity estimation of the acetone formed, is the simplest quantitative method.

**Sugars.**—**Dextrose, or glucose**, is the most important sugar met with. It is a constituent of normal urine, but the amount (0.01–0.04 per cent.) is so small that it gives no reaction with the common clinical tests. When the glucose concentration of the blood exceeds about 0.18 of one per cent. a demonstrable

amount of sugar is eliminated by the kidneys. If the normal concentration be again attained, this sugar excretion ceases; the condition is known as *temporary or intermittent glycosuria*. It may be due to a variety of causes, but can be controlled by regulating the carbohydrates of the food to the metabolic capacity of the individual. Should the attempt of the kidneys to restore a normal sugar concentration not succeed, the excretion continues and *persistent glycosuria* develops. At first, this may be a simple condition which can be controlled by regulating the diet, but later other evidence of disturbance of metabolism generally appears, and the excretion of sugar continues in spite of any limitation of carbohydrates. It is to this state that the term *diabetes mellitus* is often applied. It is obviously important to detect even intermittent traces of sugar, since in many cases they are the precursor of persistent glycosuria. The best time to collect a specimen, in suspected cases, is two or three hours after a meal rich in carbohydrates. If this is negative, the tolerance of the patient should be tested by giving 1½ gm. of dextrose per kilogram of body weight in the morning, fasting, and testing the urine at intervals for five or six hours afterwards.

**Tests.**—*Fehling's* is the classical test for sugar, but it is so unreliable that it should be abandoned and a more satisfactory, such as Benedict's, substituted. For *Benedict's test* a solution is made by dissolving 173 gm. of sodium citrate and 100 gm. of anhydrous (or 200 gm. of crystallized) sodium carbonate in about 700 c.c. of distilled water, filtering, and making up to 850 c.c. when cold; then adding 17.3 gm. of crystallized copper sulphate dissolved in 100 c.c. of distilled water, made up to 150 c.c. with constant stirring. To test for sugar, add 8 or 10 drops, not more, of the urine to 5 c.c. of Benedict's solution in a test-tube and boil for one to two minutes, then allow the tube to cool spontaneously. In the presence of sugar the entire body of the solution is filled with red, yellow, or greenish precipitate. If the quantity of sugar be small the precipitate appears only on cooling. If no sugar be present the solution remains clear, or shows a faint blue turbidity of precipitated urates. Owing to the small quantity of urine used the fallacies due to uric acid, urates, creatinin, etc., are eliminated, and, by substituting sodium carbonate for the caustic soda of Fehling's solution, the test is made more delicate. As the bulk, not the colour, of the precipitate is the

## URINE, EXAMINATION OF

important point, the test can be carried out by artificial light. Lastly, the solution does not deteriorate on keeping.

Since other sugars give a reaction, additional tests must be carried out to differentiate them. By the *fermentation test* lactose and the pentoses, which are not fermented in twelve hours at 37° C., are distinguished from dextrose, lævulose, and pseudo-lævulose, which are readily fermented. The fermentation test may be carried out with Lohenstein's, or Einhorn's, saccharimeter, or more simply as follows: A piece of compressed yeast the size of a large pea is rubbed up with 25 to 30 c.c. of the urine, which has been previously acidified with a little tartaric acid, well boiled, and cooled. A test-tube is filled to the brim with the emulsion, and then closed with a perforated cork which carries a U-shaped piece of glass tubing, stood in a beaker, and incubated at 34 to 37 C. If the urine contains a fermentable sugar, gas will accumulate in the upper end of the tube and expel a corresponding amount of urine into the beaker. To prove that the gas is carbon dioxide, the cork is removed from the end of the tube under mercury or water, and a little caustic soda introduced; the gas should be absorbed and the liquid refill the tube. Two control tests should always be carried out—(1) with normal boiled urine to which dextrose and a piece of the same yeast have been added to prove the activity of the yeast, and (2) with normal boiled urine and the yeast alone, to show that there is no gas-formation apart from fermentation.

Lactose, in the amount usually met with in the urine, does not yield an osazone with phenylhydrazine, but the pentoses do. The latter may also be recognized by the orcin test (*see p. 447*). Lævulose and pseudo-lævulose differ from dextrose in being lævorotatory and by giving a Burgundy-red colour with Seliwanow's test. (Heat in a water bath a mixture of the urine with an equal volume of a solution consisting of 0.5 grm. of resorcin, 30 c.c. of concentrated hydrochloric acid, and 30 c.c. of water.) Pseudo-lævulose gives a yellow extract and lævulose a red with Borchardt's modification of Seliwanow's test.

**Lævulose (fructose)** is said to occur in (1) alimentary lævulosuria, produced by a dose of the sugar beyond the assimilation limit: lævulosuria following a small dose is claimed to be an indication of disease of the liver; (2) pure spontaneous lævulosuria; (3) mixed mellituria, in which lævulose occurs along with

dextrose. In nearly all the cases of the last two varieties which I have investigated, I have found that the lævorotatory reducing substance was really pseudo-lævulose.

**Tests.**—Lævulose reduces Benedict's, Fehling's, and Nylander's solutions, but is lævorotatory. It is readily fermented by yeast. With phenylhydrazine it gives an osazone with the same melting-point as dextrosazone, about 200° C., but, unlike dextrose, yields a crystalline osazone, which melts at 155° C., with methyl-phenylhydrazine.

**Pseudo-lævulose (iso-glucuronic acid)** is not a sugar but a ketonic acid. Its presence in the urine appears to depend upon imperfect hepatic function. Its chief importance lies in the fact that patients whose urine contains only pseudo-lævulose may erroneously be regarded as diabetics, and consequently may be subjected to unsuitable restrictions of their diet.

**Tests.**—Pseudo-lævulose is lævorotatory, is fermented by yeast, and reduces alkaline copper solutions like lævulose, although the reduction is not quite so rapid. It gives similar osazones with phenylhydrazine and methyl-phenylhydrazine, but its para-bromphenylosazone melts at 256° C., whilst the osazone of lævulose melts at 197° C. The calcium compound of dextrose is soluble in water, but the calcium salts of both lævulose and pseudo-lævulose are insoluble. The lead salt of pseudo-lævulose is insoluble, whereas the lead compound of true lævulose is soluble.

**Lactose (milk sugar)** is mainly of interest because it may be mistaken for dextrose, or a true dextrosuria may be dismissed lightly as a physiological excretion of milk sugar. It is more commonly met with in the urine of women during the nursing period and for a short time after weaning, and has then no pathological significance. Its occurrence during pregnancy has been doubted, but I have met with a number of cases where lactose was undoubtedly present. The amount excreted rarely exceeds 1 per cent., and there is no polyuria. Alimentary lactosuria may result from the administration of lactose, and is occasionally met with after large quantities of milk have been taken, especially in patients with disorders of the gastro-intestinal tract.

**Tests.**—Lactose reduces Benedict's and Nylander's solutions, but more slowly than dextrose. Boiling with dilute mineral acids increases its reducing power. It is not fermented by yeast within twenty-four hours,

## URINE, EXAMINATION OF

although contaminating bacteria may cause slow gas-formation. The presence of lactose, or galactose, is most certainly demonstrated by the mucic-acid test.

**Pentoses** occur occasionally in the urine. No case of true or essential pentosuria had been described in the British Isles until 1920, when Howard and I reported seven which we had met with in the previous year. About 40 examples had been previously published in Germany and America. In most, the sugar has been optically inactive arabinose. Alimentary pentosuria is not very uncommon at seasons of the year when fruits containing a comparatively high percentage of pentose are being eaten. This sugar is dextrorotatory arabinose.

**Tests.**—The urine reduces Benedict's solution, but the reduction is slow, and often takes place suddenly as the solution cools. Fermentation with yeast does not prevent the reduction. Nylander's solution gives a grey precipitate. Phenylhydrazine yields an osazone which melts at about 150° C. Bial's *orcin* test is positive. Four or 5 c.c. of a reagent made by mixing 500 c.c. of fuming hydrochloric acid, sp. gr. 1.195, 1 gm. of orcin, and 25 drops of a solution of perchloride of iron, are heated to boiling and removed from the flame. The urine is immediately added drop by drop, agitating after each addition, until either the characteristic green colour develops or 1 c.c. has been added in all. It is claimed that this test does not give a reaction with glucuronic acid when properly performed.

**Glucuronic (glycuronic) acid** is not found free in the urine, but occurs in combination with lower derivatives of protein decomposition, and conjugated with certain drugs (e.g. chloral, camphor, menthol, phenol, etc.). The average daily output is 0.35-0.37 gm., chiefly in combination with phenol of intestinal origin. The excretion is increased in peritonitis, enteritis, and conditions promoting abnormal putrefactive changes in the intestine, when putrefaction occurs in other situations, in febrile and respiratory diseases, pancreatitis, and diabetes.

**Tests.**—Some of the compound glucuronates (e.g. chloral and camphor) reduce copper solution as readily as dextrose, but indoxyl-glucuronic acid only reduces after boiling for some time, while phenol-glucuronic acid does not reduce even after prolonged boiling. The glucuronates are decomposed by boiling with dilute acids, they then readily reduce Fehling's solution and give Bial's orcin test. Tollin's

test gives a characteristic reaction. The glucuronates are not fermented by yeast.

**Homogentisic acid** is another reducing substance rarely met with in the urine, and is the characteristic component in *alkaptonuria*. It is of no clinical importance, but may be mistaken for sugar. Urine containing homogentisic acid stains linen black, and on standing darkens from above downward until the whole bulk is black.

**Tests.**—Alkalis hasten the darkening whilst acids remove the colour. When a dilute solution of ferric chloride is allowed to fall into the urine drop by drop, the addition of each drop develops a deep-blue colour which lasts only a moment, and the phenomenon is repeated until oxidation is complete. Homogentisic acid is optically inactive, and is not fermented by yeast. It readily reduces alkaline copper solutions, but Nylander's solution is not affected characteristically. It forms no osazone with phenylhydrazine.

**Estimation of sugar.**—The estimation of sugar in the urine is most accurately carried out by the method I described in the *Lancet* in 1917 and 1919 (April 21st, 1917, p. 613, and May 31st, 1919, p. 939), which may also be used for blood sugar estimation, but for clinical work Benedict's gives satisfactory results, and is to be preferred to the Fehling process. The quantitative solution is *not* the same as that used for qualitative work. Dissolve 200 gm. of sodium citrate, 200 gm. of crystallized (or 100 gm. of anhydrous) sodium carbonate, and 125 gm. of potassium sulphocyanide in about 800 c.c. of distilled water, cool, and filter. Dissolve 18 gm. of pure, air-dried crystalline copper sulphate in about 100 c.c. of distilled water and pour into the former solution with constant stirring. Add 5 c.c. of a 5-per-cent. solution of potassium ferrocyanide and enough distilled water to make up to 1,000 c.c. This solution keeps indefinitely. To carry out the estimation, fix a small flask so that its contents can be kept briskly boiling with a small flame, introduce 3 to 4 gm. of anhydrous sodium carbonate (about an inch in a dry test-tube), add 20 c.c. of the reagent, and heat until the carbonate is dissolved. Run in the urine from a graduated burette until a milky-white precipitate is formed and the blue colour of the solution is perceptibly lessened, then more slowly until the last trace of blue has disappeared, boiling vigorously all the time. Toward the end the urine should be added a drop or two at a time with 30 seconds interval



## URINE, EXAMINATION OF

between each addition. If less than 5 c.c. are used, dilute the urine so that about 10 c.c. will be necessary to complete the reduction, and repeat the estimation. If the solution becomes too concentrated, distilled water may be added. Should excessive bumping occur, a small quantity of powdered pumice stone may be added. Each 25 c.c. of Benedict's solution is reduced by 0.05 gm. of dextrose, 0.053 gm. of lævulose, and 0.0676 gm. of lactose. If  $y$  = the dilution of the urine and  $x$  = the quantity used, then  $\frac{0.05 \times 100 \times y}{x}$  = the percentage of dextrose in the original urine.

If lævulose or lactose be present in pure solution the factors given are substituted for 0.05 gm. When the urine contains a fermentable and an unfermentable sugar, the proportions can be determined by the difference between the percentages shown by a saccharimeter (Lohenstein's is the best) and by titration. If a mixture of dextrose and lævulose be present, the percentage of each sugar may be ascertained by comparing the readings obtained by titration and on examination with the polariscope. As the lævorotatory reducing body is usually pseudo-lævulose and its calcium salt is insoluble, Howard and I have devised a method of estimation in which advantage is taken of this fact. Mix 10 c.c. of the urine with 1 c.c. of 30-per-cent. sodium hydrate and 4 c.c. of 10-per-cent. calcium chloride solution, centrifuge for three minutes and filter off the supernatant liquid. The sugar in the filtrate is then estimated. The difference between this reading (allowing for the dilution) and that for the untreated urine shows the percentage of pseudo-lævulose. Pentoses cannot be estimated accurately by reduction methods; Jolles' distillation process gives the best results.

**Dextrins** occur in normal urine in traces. In conditions where carbohydrate metabolism is being imperfectly carried out the excretion is increased and may precede the appearance of sugar in the urine by a considerable period. This is the basis of the so-called "*pancreatic reaction*" in the urine. The presence of dextrin is shown by boiling the urine with hydrochloric acid to split off the pentose (xylose) it contains, neutralizing with lead carbonate, removing the glucuronic acid with basic lead acetate, and precipitating the pentose as yellow osazone crystals. This reaction is not pathognomonic of disease of the pancreas, but, as affections of the pancreas are the commonest

cause of a disturbance of carbohydrate metabolism, a positive reaction is helpful in diagnosis. In an improved quantitative method, which can be used in diabetes, the urine is hydrolyzed, neutralized, and the glucuronic acid removed as before, but the sugar is precipitated from the filtrate as a lead salt by the addition of ammonia. The precipitate is then washed with water, dissolved in hydrochloric acid, and distilled with steam. The distillate containing the furfural formed by the action of the acid on the pentose is collected, treated with a solution of sodium sulphite, and the excess of sulphite titrated with centinormal iodine. The difference between this reading and that given by the same amount of sulphite added to distilled water is termed the *iodine coefficient* of the urine when it is worked out for the twenty-four hours' excretion. The iodine coefficient of normal urine is nil. Figures ranging from 50 to 500 are obtained when carbohydrate metabolism is defective owing to disease of the pancreas. When sugar appears in the urine, the iodine coefficient falls, to rise again when the sugar diminishes or disappears. The iodine coefficient and the sugar excretion are generally of inverse dimensions, but in severe cases of diabetes they may run together.

The **total nitrogen output** in the urine of a normal adult, on an average mixed diet, ranges from 10 to 16 gm. in the twenty-four hours. It is the best index we have of protein metabolism. The proportion between the intake and the output of nitrogen is termed "the nitrogen balance." When they are equal the individual is said to be in "nitrogenous equilibrium." The total nitrogen is increased by conditions which raise protein metabolism, such as a meat diet, exercise, and hot baths. Pathologically, it is increased by acute infections, by poisoning by various metals, during the absorption of exudates and transudates, in oxygen starvation, by severe hæmorrhage, in malnutrition, diabetes mellitus, malignant disease, chronic infections, pernicious anæmia, leukæmia, scurvy, and exophthalmic goitre. A diet rich in carbohydrates reduces the nitrogen excretion below the fasting level. A decrease takes place during convalescence from acute diseases, during the formation of exudates and transudates, from sweating, vomiting, and diarrhœa, in acute yellow atrophy of the liver, in myxœdema, and in nephritis. The total nitrogen of the urine is estimated by Kjeldahl's method, or some modification of it.

## URINE, EXAMINATION OF

Rona and Ottenberg's modification or Folin's micro-chemical method is the most convenient.

**Urea** is the chief nitrogenous constituent of the urine. The amount excreted by a normal person on an average diet is about 0.4 grm. per kg. of body weight, and varies from 1 to 3 per cent., with a total of 20 to 40 grm. a day. Less is passed in old age and by fat subjects. Since about 90 per cent. of the nitrogen of the food leaves the body as urea, a protein-poor diet causes a fall in the urea excretion, whilst a meat diet increases it. As a rule, the urea and the total nitrogen excretions run parallel. Part of the nitrogen which should be passed as urea is sometimes excreted in other forms, notably as ammonia. This occurs in diseases of the liver and in conditions where carbohydrate metabolism or absorption is interfered with. In many chronic destructive diseases of the kidneys the percentage of urea is much below normal, but the total amount may not be greatly reduced. The excretion of urea is diminished in acute and subacute nephritis with increasing dropsy, and in the last stages of chronic interstitial nephritis, also in renal congestion and other forms of nephritis, especially before the onset of uræmia. The passage of only a small amount of urea is not of itself an infallible sign of uræmic poisoning. The persistent excretion of a subnormal amount of urea is suggestive of renal insufficiency, particularly in association with chronic interstitial nephritis. The hypobromite method for estimating urea is most unreliable. The time required for Marshall's urease process is not much greater and the results are accurate. A small volume of the urine is incubated with Soya bean meal, or an extract of urease made from it, and the urea is converted into ammonium carbonate. By adding an excess of caustic alkali and aerating the mixture, the ammonia is driven off and carried over into a known volume of standard acid, which by titration with standard alkali shows the amount of ammonia formed. Or the ammonia may be estimated by the Folin-Denis permutit process. The amount of ammonia being known, the percentage of urea can be calculated.

**Ammonia** is the most important nitrogenous constituent of the urine next to urea. It occurs combined with acids, and represents from 3.5 to 7 per cent. of the total nitrogen. A normal person on a mixed diet passes from 0.3 to 1.2 grm. of ammonia in twenty-four hours. The ratio of urea to ammonia is normally about

30 to 1. Ammonia is increased in the urine by the administration of mineral acids and organic acids which cannot be further oxidized in the body, also by the formation of an excess of acids in the metabolic processes. The latter are increased by a rich protein diet, by violent exercise, oxygen starvation, in fevers, in diabetes mellitus, and in pernicious vomiting of pregnancy. Destructive diseases of the liver are associated with a rise in the ammonia of the urine, but the diagnostic value of such an increase is not so important as was at one time believed. The chief value of ammonia estimations lies in the indications they give as to the presence and extent of the formation of abnormal acids in conditions of perverted metabolism, and particularly diabetes mellitus. The relation existing between the ammonia-nitrogen and the total nitrogen, also between the ammonia-nitrogen and the urea-nitrogen, are more important than the absolute values.

**Estimation.**—Two methods are commonly employed for the estimation of ammonia. By the "formalin" process the amino-acids are determined along with the ammonia; by the Folin-Denis permutit process (*Journ. Brit. Chem.*, xxix, 1917, p. 333) the ammonia only is estimated. So long as the amino-acids do not exceed the normal traces the two methods give concordant results, but when they are abnormally increased the difference between the readings is a measure of the amino-acid content of the urine. It is consequently advisable to employ both methods and compare the results, especially as the amino-acid content of the urine is of considerable diagnostic value. As a rule, an ammonia value of over 2 grm. in the twenty-four hours is an unfavourable sign in diabetes, and over 5 grm. suggests that coma is imminent. A high ammonia-nitrogen coefficient and a low urea-ammonia ratio are still more important indications of serious acidosis. It is hardly necessary to point out that fresh urine is necessary for the estimation of ammonia.

**Amino-acids** occur normally in small quantities, accounting for 1.5-3.6 per cent. of the total nitrogen. An abnormal amino-acid content is one of the most important indications of a functional insufficiency of the liver. When extensive destruction of the parenchyma occurs, amino-acids are often present in such quantities that the less soluble, notably tyrosine, crystallize out. An excess of amino-acid nitrogen is met with in gout, the toxæmias of pregnancy, eclampsia, tuberculosis, typhoid

## URINE, EXAMINATION OF

fever and other intestinal diseases, smallpox, pernicious anæmia, leukæmia, and some cases of diabetes mellitus. In simple glycosuria the urine is free from an excess of amino-acid, unless there is an associated gouty condition or serious hepatic derangement.

**Uric acid** occurs free in minute quantities, but at times an abundant sediment of "red sand," insoluble on warming, may form, owing to its being liberated from its combinations through changes in the reaction of the urine and other causes. Urates may also be deposited from concentrated urines on cooling, forming a dirty-white or brick-dust sediment (soluble on warming). The uric acid of the urine is of twofold origin; one (the "endogenous" fraction) is derived from the purin bodies formed in the metabolism of tissue proteins, the other (the "exogenous" fraction) comes from the purins in the food. On an average diet, the uric acid, calculated as the free acid, varies between 0.2 and 1.2 grm. The excretion in infants and young children is higher in proportion to their weight. In adults the ratio of the nitrogen of the uric acid to the nitrogen of the urea is about 1 to 50-70, but in the new-born it is 1 to 13-14. Physiologically, the excretion of uric acid is increased by raising the nuclein content of the diet by meat broths and drinks containing caffeine, and by certain drugs, etc. There is considerable diversity of opinion concerning the excretion of uric acid under pathological conditions, owing chiefly to the difference between endogenous and exogenous uric acid not having been allowed for by many observers. The increase met with in fevers, leukæmia, and the resolving stage of pneumonia is no doubt endogenous. The great excess met with in diabetes mellitus is often due to the diet, but in severe cases is contributed to by the excessive tissue waste. An increase is said to occur in cirrhosis of the liver, splenic diseases, malaria, scurvy, dyspnoic disorders, and diseases interfering with the circulation, in association with abdominal tumours, especially in women, in gastro-intestinal toxæmia, and neurosis. The excretion of uric acid is generally increased in malignant disease, while the output of phosphates is diminished; a study of the ratio between the two is consequently of assistance in diagnosis. A ratio below 1:4 is suggestive of cancer, and a ratio below 1:3 is strong evidence when confirmed by other signs and symptoms. In chronic gout the uric-acid excretion tends to be below the normal. For

one to three days before each acute attack it decreases considerably, rising with the onset of the attack, and falling again in the course of a few days. The uric-acid content of the urine is diminished by a purin-poor diet, by lead poisoning, by advanced renal disease, after large doses of quinine, and in certain chronic diseases.

**Tests.**—The most important chemical property of uric acid is its power of reducing Fehling's solution, but it does not reduce Benedict's solution or alkaline solutions of bismuth.

**Estimation.**—The Ludwig-Salkowski method is the most accurate for estimating uric acid, but the more rapid processes devised by Folin and Benedict and Hitchcock are now generally employed in clinical work. Ruhemann's ureometer is easy to use, but its accuracy is doubtful. Some indication of the amount of uric acid present in non-albuminous urines is given by Heller's test, a cloudy ring above the junction within five minutes indicating an excess.

**Other purin bases** (alloxuric, xanthin, or nuclein bases).—On a mixed diet from 16 to 60 mg. are excreted in the twenty-four hours. There is an increase during severe muscular exercise, in fevers, leukæmia, enteritis, tuberculosis, and as a result of X-ray treatment.

**Estimation.**—The output may be estimated by precipitating the total purins with ammoniacal silver nitrate, determining the nitrogen in the precipitate by Kjeldahl's method, and subtracting the uric-acid nitrogen from the result, or by one of the purinometers on the market, care being taken to have the patient on a purin-free diet to exclude exogenous purins.

**Creatinin** occurs normally to the extent of 0.6-1.3 grm. in the twenty-four hours. It is stated to be an index of muscle katabolism, and the creatinin coefficient (mg. creatinin-nitrogen per kilo of body-weight) is regarded as a measure of the muscular efficiency of the individual. For men the coefficient normally ranges between 8 and 11. The excretion of creatinin is increased by a meat diet, in complete starvation, in fevers, and by exercise. A marked increase is frequently met with in diabetes. In most diseases of the liver there is a decreased excretion, but in hepatic carcinoma the creatinin is increased and accompanied by creatin. Creatinin reduces Fehling's solution, but not Benedict's nor Nylander's solution. It is readily estimated by Folin's colorimetric method. *Creatin* is a normal constituent of children's urine, but in adults it is met with

## URINE, EXAMINATION OF

only after a meat diet. Pathologically, it occurs in starvation and fevers, severe diabetes, and acromegaly.

**Albumin** is never found in the urine in health. The occurrence of heat-coagulable protein in the urine is termed albuminuria, no matter whether the protein is serum-albumin or serum-globulin. As a rule both are present. *True albuminuria* may be brought about by alterations in the blood-pressure in the kidneys, by changes in the composition of the blood, or by alteration in the structure of the kidneys. In *false or accidental albuminuria* the albumin does not come from the kidneys, but gains access lower down the uro-genital tract.

**Tests.**—For all albumin tests the urine should be clear and as fresh as possible. In addition to a twenty-four hours' collection, a fresh night and morning sample should be examined in doubtful cases. Much difference of opinion exists as to the best tests. My own procedure is to employ salicyl-sulphonic acid first. If no reaction is obtained the investigation is not carried further, and if a precipitate forms the turbid fluid is boiled to detect the presence of albumoses. A second sample is then examined with Heller's cold nitric-acid test. If this gives no reaction in three minutes, any albumin that is present is considered to be of no clinical importance. A nucleo-protein ring a centimetre or so above the line of contact may afford an explanation of a slight reaction with salicyl-sulphonic acid. Finally, in some instances, resort is had to Purdy's test. The *salicyl-sulphonic acid test* is convenient and easy. A few drops of a saturated solution, or a few crystals of the solid, are added to the urine and shaken. In the presence of albumin a white precipitate forms. The test is very delicate, showing 1 in 20,000 immediately, and 1 in 100,000 in two or three minutes. Albumoses also give a precipitate, but it dissolves on heating, to reappear on cooling. The nucleo-protein ("mucus") present in normal urines may give a faint haze. *Heller's "ring" test*, properly performed, is very reliable. It is fairly sensitive, showing 1 in 12,000 immediately, and 1 in 30,000 on standing for three minutes, as a white ring at the junction of the fluids, when the urine is gently run on to an inch layer of nitric acid in a test-tube. The superimposition is most evenly effected by filtering the urine so that the filtrate runs down the side of the tube. The ring is best seen against a black background. It is essential that the urine should be run on

to the acid and not the reverse. The nitric acid must be pure. The most common cause of a doubtful reaction is nucleo-protein; in this case, however, the precipitate does not appear as a ring at the junction, but is seen as a haze 0.5 to 1.0 cm. above the line of contact. In concentrated urines a deposit of urea nitrate may form; it appears slowly, is obviously crystalline, and is prevented by diluting with water. Urates may give rise to a diffuse opacity, which is often brown, spreads downwards, and disappears on warming or diluting the specimen. After the administration of various resinous drugs, a uniform turbidity which is soluble in alcohol, may be seen.

The tests for albumin which depend on its coagulation by heat are those most commonly employed, but they have many drawbacks. The modification which meets these difficulties most satisfactorily is known as *Purdy's test*. To 10 c.c. of the urine add one-sixth of its volume of a clear saturated solution of common salt, and shake. Acidify the mixture with acetic acid and heat the upper part. A cloud appears in the heated portion if albumin be present, and on boiling the precipitate collects in flocculi. If there be much albumin the precipitate appears in the cold. Albumoses give a cloud on warming, which disappears when the liquid is boiled, to reappear as it cools. In a mixture of albumoses and albumin the former may be demonstrated by filtering the boiling fluid and examining the filtrate. The test is as sensitive as the ordinary heat test, but avoids the nucleo-protein difficulty; moreover, salt and vinegar (6 per cent. of acetic acid) are always at hand, and the test can be carried out in a spoon over a candle-flame.

**Estimation.** Esbach's method is commonly employed for the estimation of albumin, but the figures obtained are misleading and inconsistent. Better results are given by *Goodman and Stern's method*. Five c.c. of a solution consisting of phosphotungstic acid (1.5 gm.), concentrated hydrochloric acid (5 c.c.), and alcohol (95 per cent.) to 100 c.c. are placed in a clean, dry test-tube, and the filtered urine is added drop by drop from a graduated pipette, shaking between each addition, until a faint white cloudiness is seen. The reading on the pipette is then taken. The quantity of urine used contains  $\frac{1}{10}$  mg. (0.0001 gm.) of albumin, and from this the percentage can be calculated. As a rule, it is advisable to dilute the urine, making an allowance in the final

## URINE, EXAMINATION OF

calculation. If  $x$  = the quantity of urine used, and  $y$  = the dilution, then  $y \times 0.0001 \times 100 = x$  = grm. of albumin per cent.

**Albumoses** may be found in the urine when tissues or exudates are being autolysed and absorbed. They have been met with in suppuration, in resolving pneumonia, in involution of the uterus, with malignant disease, febrile diseases, acute yellow atrophy, phosphorus poisoning, leukæmia, eclampsia, pulmonary tuberculosis, and after tuberculin injections. They are not usually found in nephritis, except with the chronic parenchymatous variety.

**Tests.**—Albumoses may be recognized by the tests mentioned under Albumin.

The so-called **Bence-Jones albumose** or **protein**, which appears in the urine of patients with multiple bone-marrow tumours, is not a true albumose, but is more closely related to the simple proteins. Its coagulation temperature is low, 49–60° C., but is modified by the amount of salts and urea. In many cases the coagulum is re-dissolved at a higher temperature, to reappear on cooling, but this is not invariable. A precipitate is formed by strong (25 per cent.) nitric acid, which disappears on heating and reappears on cooling. Strong hydrochloric acid causes a dense precipitate which is typical. No precipitate is caused by acetic acid, even in excess, and the heat coagulum is dissolved on adding this acid. It is precipitated by alcohol and after standing loses its solubility, thus differing from true albumose.

**Urochrome** is the chief pigment of normal urine. It is increased in acute febrile disorders, in extensive diseases of the liver, chronic diseases of the heart and lungs, chronic renal hyperæmia, and all conditions with increased destruction of erythrocytes. Its chief chemical characteristic is its great reducing properties.

**Urobilin (hydrobilirubin)** is met with in fresh urine as a chromogen, urobilinogen. This is converted into urobilin by the action of light and oxygen. The quantity varies in health from 30 to 120 mg. in twenty-four hours. Its presence in excess is often an evidence of hepatic insufficiency, and is found in cirrhosis, carcinoma, and trauma of the liver, in association with gall-stones in the common bile-duct, in diseases of the bile-ducts, duodenal catarrhs, and typhoid fever. An excess also occurs in conditions accompanied by abnormal blood-destruction, in poisoning by certain drugs, and during the absorption of blood-clots.

**Tests.**—Urobilinogen is detected by Ehrlich's aldehyde reaction. With the spectroscope, urobilin shows a band between the green and the blue (B and F). Chemically, it is detected by the *zinc acetate test*. Mix the urine with an equal bulk of a saturated solution of zinc acetate in alcohol, shake, and filter through a dry filter-paper. On standing overnight, a green fluorescence is seen when the solution is examined by reflected light against a black background. *Ehrlich's diazo reaction* gives with normal urine an orange colour, but in typhoid fever, measles, tuberculosis, septicæmia, and some cases of malignant disease the colour varies from a light carmine to a deep red. Two solutions, which are kept separately in well-stoppered amber glass bottles, are required: (A) sulphanilic acid 2 grm., hydrochloric acid (1.19) 50 c.c., distilled water 1,000 c.c.; (B) sodium nitrite 1 grm., distilled water 200 c.c. The second should be made fresh. Add 50 c.c. of solution (A) to 1 c.c. of solution (B), and to 10 c.c. of the mixture add an equal volume of fresh urine. Shake well and overlay with 3 c.c. of ammonia. A positive result is shown by the appearance of a red ring at the junction. On shaking, the entire liquid becomes pink and the foam is pink or rose-coloured. If the mixture be poured into a dish of water the liquid becomes pink, but with normal urines is yellow. The only colours of diagnostic importance are a carmine or crimson in the liquid and the pink or rose foam on shaking.

**Hæmatoporphyrin** is present in traces in normal urine. It is increased in acute infections, in poisoning by sulphonal, trional, or tetronal, in functional disorders of the ductless glands, in primary anæmias, cirrhosis of the liver, gout, and lead poisoning. Urine containing an appreciable amount of the substance is reddish-brown in thin layers, but by reflected light may be red, port-wine colour, or almost black. On standing, the colour darkens.

**Test.**—To identify the pigment it should be isolated by precipitating with barium nitrate and baryta water, washing the sediment, and extracting with acid alcohol. On examining the extract with the spectroscope a narrow band in the yellow, and a broader band between the yellow and the green, are seen.

**Hæmoglobin** is found in the urine only under pathological conditions. It is met with as the result of a destruction of red blood-corpuscles, or following hæmorrhage into the

## URINE, EXAMINATION OF

genito-urinary tract. The former condition is known as hæmoglobinuria, the latter as hæmaturia.

*Hæmoglobinuria* occurs when one-sixth of the total hæmoglobin of the blood is set free. This may result from the action of poisons, in fevers as in blackwater fever, from severe burns, from exposure to cold, from transfusion of foreign serum, during pregnancy, as an epidemic fever in the new-born (epidemic hæmoglobinuria), in nephritis, and after severe intra-abdominal hæmorrhage. The urine may be clear, but is usually cloudy from casts and masses of pigment. A few red cells may be seen, but not sufficient to account for the amount of hæmoglobin. As erythrocytes are apt to break up quickly, it is important, however, that urine should be examined while fresh.

**Tests.**—The *benzidine test* for hæmoglobin is the most delicate. To 10 c.c. of the urine add 1 c.c. of glacial acetic acid and shake. Then add 3–4 c.c. of ether and shake again. Stand for a few minutes, add 5–10 drops of absolute alcohol and shake gently. Remove the ether with a pipette and add to it a fresh solution of 0.5 gm. of Merck's pure benzidine in sufficient glacial acetic acid (with which 2 or 3 c.c. of a 3-per-cent. solution of hydrogen peroxide have been mixed) to dissolve it. Shake the mixture well. A green or blue colour is seen according to the amount of hæmoglobin. The *guaiac test* is not so sensitive, and reacts with pus, bile, and other accidental constituents. Prepare a fresh alcoholic solution of guaiacum resin (1:5), stand for a few minutes, and filter. Mix equal volumes of the filtrate and commercial hydrogen peroxide, or ozonized turpentine, and shake. Run in an equal volume of the urine carefully. In the presence of hæmoglobin a blue ring appears at the junction, and on shaking, the whole liquid turns blue. Warning the mixture does not affect the colour if it be due to blood, but the blue due to pus disappears. Examined with the *spectroscope*, the urine usually shows the mixed spectrum of oxyhæmoglobin, reduced hæmoglobin, and methæmoglobin. In the hæmoglobinuria of nephritis the dark band in the red, between c and d, of methæmoglobin is generally most conspicuous, but in hæmaturia where the blood is fresh the two bands of oxyhæmoglobin, between d and e, predominate. The spectrum may be confused by other bodies which give bands or darken the field, such as hæmatin, bile, or urobilin. In any

case, the nature of the spectrum should be confirmed by converting the oxyhæmoglobin into reduced hæmoglobin by adding ammonium sulphide.

In *hæmaturia* blood is found chemically, the hæmoglobin tests given above being positive, and the microscope shows many red blood-corpuscles as well. The colour ranges from light smoky to bright red if hæmoglobin predominate, and to dark brownish-red if methæmoglobin be mainly present. Hæmaturia may be due to blood diseases and malignant forms of infection, to kidney diseases, disease of the lower urinary passages, injuries of the urinary tract, and to "essential renal hæmaturia." It may be of menstrual origin, or arise from fistulous openings into the urinary tract. Renal blood and urine are well mixed, so that separately voided quantities contain the same amount of blood, clots are not common, and on washing out the bladder the washings finally come away clear. The discovery of tube-casts or shadow-corpuscles in the fresh urine, and the absence of pain, excessive frequency, or straining on micturition, point to a renal origin.

**Bile pigments** occur in the urine only pathologically. The condition is known as choloria, and is always associated, sooner or later, with jaundice. In toxic ("hæmatogenous") jaundice bile pigments may appear in the urine several days before the conjunctivæ and skin are stained. In freshly passed urine bilirubin occurs alone, but on standing, oxidation products, notably biliverdin, are formed. The colour of urine containing bile varies therefore from bright yellow to greenish brown or almost black, but a light-coloured urine may contain a considerable amount of bile. The most characteristic feature is the yellow foam produced on shaking. The urine stains filter-paper yellow, the sediment is yellow, and, microscopically, any epithelial cells and casts are seen to be of an intense golden yellow colour.

**Tests.**—Heller's cold nitric acid test shows the nucleo-protein ring to a marked degree, and a colour zone is seen. If nitric acid containing nitrous acid be used for the test a range of colours from green above, through blue, violet, and red, to yellow below, develops (*Gmelin's test*). Green is the essential colour of the test. If much indican be present a deceptive greenish blue may appear. It is best to use fresh urine and to dilute it to a specific gravity of 1.005, for then only does the

## URINE, EXAMINATION OF

green appear. The colours may be masked if albumin be present. *Rosenbach's modification* is more sensitive, and avoids the albumin difficulty. A large quantity of urine acidified with hydrochloric acid is filtered through a white filter-paper. The paper is then partly dried and a single drop of yellow nitric acid dropped upon it. Rings of colour are seen with the green outside. Some prefer the *iodine test*, as it avoids the indican fallacy, but it occasionally gives a reaction with normal urines. The urine is acidified with acetic acid, and a 1-per-cent. solution of iodine in alcohol is run on to the surface. If bile be present, a green colour appears at the line of contact at once, or within one minute, and the whole fluid turns green on shaking.

**Bile acids** occur in the urine in some cases of jaundice, but not in all. It was formerly supposed that their presence excluded toxicæmic jaundice, but they may be present in traces and absent in "obstructive" jaundice, especially when it is of old standing.

**Test.**—*Hey's test* is the simplest, and is given by no other substance. The urine is cooled to 17° C., or lower, and a little finely powdered sulphur sprinkled on the surface. If the sulphur sink at once, it indicates 1:10,000 of bile acids; if it sink after shaking and waiting one minute, 1:40,000.

**Ferments** having the power of digesting fibrin, of inverting starch, and of coagulating milk are found in the urine in health. Lipase does not occur normally, but in acute diseases of the pancreas it can often be demonstrated. Variations in the amount of urinary pepsin are not of much diagnostic value, although it is said that a large amount, with a deficiency of gastric pepsin, points to gastric carcinoma. Variations in the diastatic power of the urine may be due to alterations in the diastase content of the blood or to changes in the permeability of the kidneys. As a rule, a diminution is found in renal diseases, except in some cases of marked albuminuria. In pancreatic affections the urine has a high diastatic value, provided that no renal disease exists.

**Estimation.**—*Wohlgemuth's method* of estimating the diastatic power is usually employed. Into a series of small test-tubes diminishing quantities of the urine from 0.5 c.c. to 0.01 c.c. are placed, and to each is added 2 c.c. of a 0.1-per-cent. solution of starch. They are then kept at 37° C. for half an hour. Four drops of centinormal iodine solution are added to each tube, starting with that containing

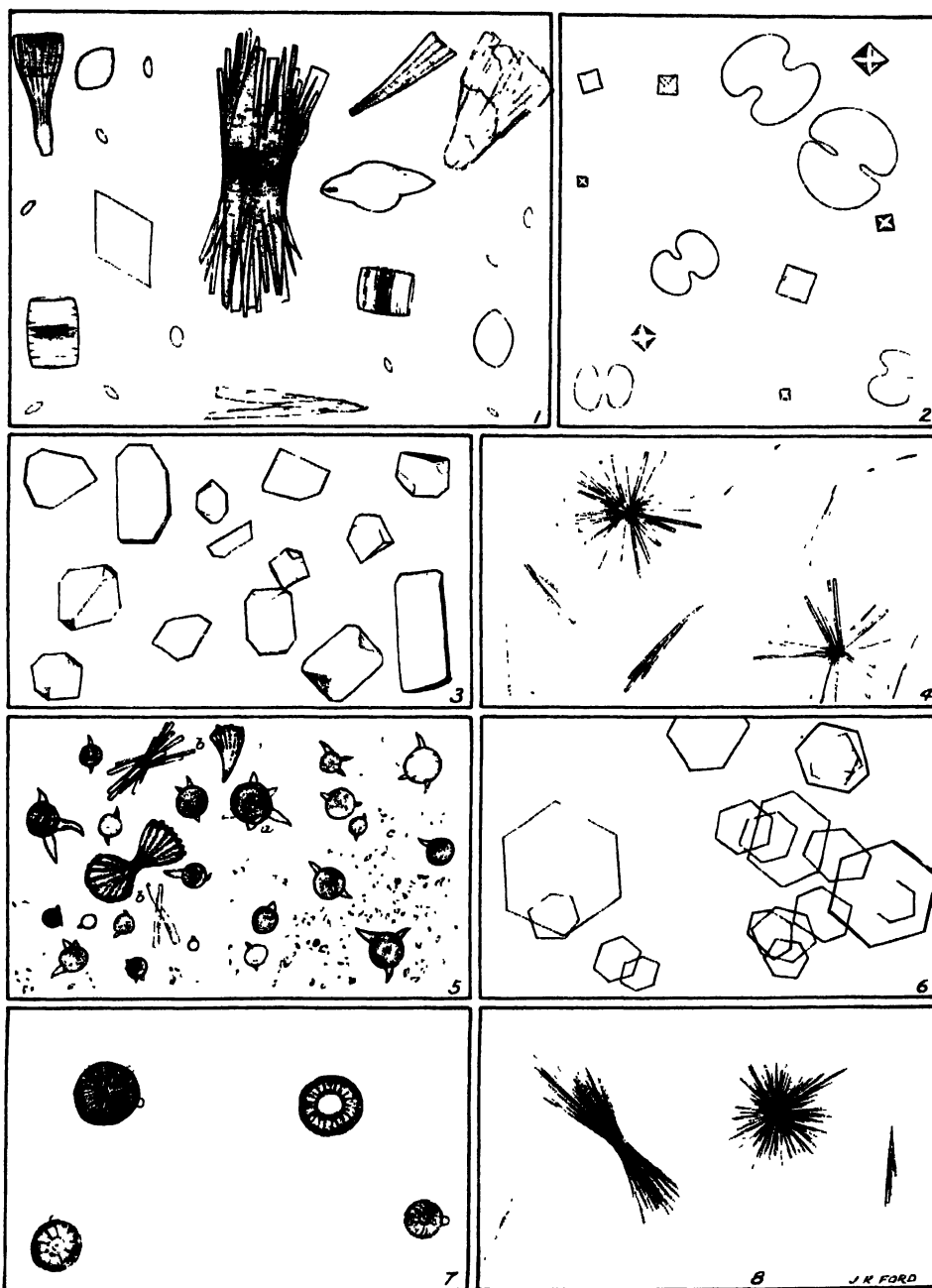
0.5 c.c. of urine. The tube previous to that which first gives a distinct blue colour is considered to contain the amount of urine capable of inverting the added starch. To determine the number of units present, the amount of starch (2 c.c.) is divided by the quantity of urine present in the tube. In health the number of units passed varies from 6.6 to 33.3, with an average of 10 to 20, for the mixed twenty-four hours' urine. Abnormal constituents, except blood, do not affect the value. Readings up to 100 may be met with in acute infectious conditions, but values above this are found only in pancreatic disease.

**Microscopical examination.**—Fresh urine should be used, and a centrifuge is desirable for separating the sediment.

**A. Unorganized sediments** (Plate 39).—It is convenient to classify these according to the reaction of the urine in which they are usually found, but many of them are met with under other conditions.

The MORE COMMON VARIETIES are: (A) *In acid urine.* (1) *Uric acid* crystals: large light-yellow to reddish-brown rhombic prisms, rosettes, dumb-bells, or whetstone shapes, soluble in sodium hydroxide and re-precipitated by hydrochloric acid. (2) *Urates of sodium and potassium*: amorphous, soluble on warming and in acetic acid. (3) *Calcium oxalate* crystals: colourless, highly refractile octahedra ("envelope shape"), occasionally dumb-bells, or discs, often very minute, insoluble in acetic acid, easily soluble in hydrochloric acid. (B) *In amphoteric or alkaline urine.* (1) *Ammonium-magnesium ("triple") phosphate* crystals: colourless triangular prisms ("coffin lid"), often very irregular, sometimes in squares like calcium oxalate, easily soluble in acetic acid, insoluble in alkalis. When these crystals are found the urine is often ammoniacal. (2) *Acid calcium (stellar) phosphate* crystals: colourless wedge-shaped rods, usually grouped in rosettes, soluble in weak acetic acid and 20-per-cent. ammonium carbonate solution. (3) *Earthy phosphates*: minute amorphous white granules, soluble in dilute acetic acid but not dissolved by alkalis or by warming.

The LESS COMMON VARIETIES of unorganized sediments are: *Acid sodium urate*, in amphoteric urine undergoing ammoniacal decomposition, as fan-shaped clusters or prismatic needles. *Ammonium urate*, in acid urine of children and alkaline ammoniacal urines of adults, as dark-brown spheres covered with fine spicules ("thorn-apple"), soluble on warming in acetic



1, Uric-acid crystals. 2, Calcium-oxalate crystals. 3, Triple phosphate crystals. 4, Stellar phosphate crystals. 5, (a) Crystalline ammonium urate ("thorn-apple"); (b) crystalline sodium urate; (c) amorphous urates. 6, Cystin plates. 7, Leucin crystals. 8, Tyrosin crystals. ( $\frac{1}{4}$  objective.)

PLATE 39.—UNORGANIZED DEPOSITS IN URINE.





## URINE, EXAMINATION OF

acid. *Calcium carbonate*: small granules, spheres, or dumb-bells with radiating structure, soluble in dilute mineral acid with liberation of gas. *Calcium sulphate*, in concentrated urine of high acidity, as long thin colourless needles or plates, insoluble in acetic acid and soluble in hydrochloric acid with difficulty. *Magnesium phosphate*, in feebly acid or alkaline urine, as highly refractile long rhombic plates with bevelled edges, soluble in acetic acid and slowly soluble in 20-per-cent. ammonium carbonate. *Cystin*, in pale urine which on standing gives off an odour of sulphuretted hydrogen, as colourless hexagonal plates, insoluble in acetic acid, soluble in hydrochloric acid, soluble in ammonia. *Creatinin* in the sediment from acid urines, especially after severe exercise, as colourless or light-green crystals resembling uric acid in shape but showing a radiating striation under high powers. *Cholesterin*: thin colourless rhombic plates, often with broken angles or sides. Dilute sulphuric acid followed by iodine solution gives a play of colours, violet, blue-green, and blue. *Hippuric acid*: colourless, or pigmented, four-sided semi-transparent rods and prisms, or clusters of fine needles, soluble in ammonia, insoluble in acetic acid. *Tyrosin*: sheaves or rosettes of fine dark-greenish yellow needles, insoluble in acetic acid, soluble in dilute mineral acids and alkalis. *Leucin* is not found as a spontaneous deposit, but separates out, with tyrosin, as yellow spherical drops showing radial and concentric striations, soluble in acids and alkalis. *Hæmatoidin* (bilirubin): minute red-brown needles or rhombic tablets, in hæmorrhagic nephritis, jaundice, acute yellow atrophy, etc. *Indigo*: amorphous flakes or blue stellate needles or rhombs, soluble in chloroform. They are generally found in alkaline decomposing urine from cases of jaundice, abscess of the liver, or intestinal obstruction.

**B. Organized sediments** (Plate 40).—*Mucus* is present in urine, partly dissolved and partly as a cloudy turbidity which sinks on standing; it is transparent and is rendered visible by embedded extraneous elements. Acetic acid makes it more distinct. *Leucocytes* exist normally in small numbers in the mucous deposit. They are distinguished from epithelial cells by the deep-brown colour they give with iodo-potassium iodide solution. When present in large numbers they constitute *pus*. In acid or amphoteric urines their form is well preserved, but in alkaline urines they swell up to a shapeless mass. With strong sodium hydrate, *pus*

forms a tough slimy mass (Donne's test) and gives a blue colour with tincture of guaiac and acetic acid (Vitali's test). Urine containing *pus* always contains albumin. *Erythrocytes* retain their normal shape and colour when freshly shed, but are generally swollen and pale ("shadow corpuscles") or may be crenated. They are readily soluble in acetic acid. Roundish *epithelial cells* generally come from the urinary tubules; large flat polygonal cells from the superficial layers of the urinary passages or vagina; columnar, conical, or caudate cells from deeper layers of the urinary passages.

*Casts* are cylindrical structures with sharply defined parallel sides and rounded ends. They are of renal origin and owe their form to the renal tubules. As a rule they are more abundant in urine passed in the evening than in the morning. The following types are distinguished: (1) Epithelial casts, composed of epithelial cells from the urinary tubules, usually in a state of granular or fatty degeneration. (2) Granular casts are formed when the degeneration is more advanced and the outline of the cells becomes lost. The granules consist of albumin or fat, and may be large or small, so that a distinction is made between "finely" and "coarsely" granular casts. If the granules consist of albumin they are made more distinct by acetic acid. If they consist mainly of fat they attract attention by their glistening appearance. (3) Hyaline casts have a pale, homogeneous, transparent basic substance, but the margins are always distinct. Their detection is simplified by the addition of a few drops of iodo-potassium iodine solution. (4) Waxy casts resemble hyaline casts, but are larger, broader, and tougher. They are yellowish in colour and moderately refractile. (5) Blood-casts consist of red and white corpuscles cemented together with fibrin, and are a result of hæmorrhage into the renal tubules. *Cylindroids*, which are found in both normal and pathological urine, must be distinguished from true casts. They consist of mucus, and differ from hyaline casts in being distinctly striated longitudinally and in having tapering ends.

*Clumps of bacteria* ("bacterial casts") resembling granular casts are occasionally found. *Urinary filaments* ("urethral threads") are much larger than casts, and consist of a homogeneous transparent basic substance in which is a varying quantity of epithelial cells, leucocytes, and, often, amorphous or crystalline salts. They are products of the mucoid or purulent secretion of the urethra and genital

## URINE, EXTRAVASATION OF

glands. *Fragments of tissue* are recognized by their histological structure. *Spermatozoa* are frequently present in the sediment, sometimes singly, occasionally in large numbers. Of the *animal parasites*, the echinococcus and the ova of the bilharzia are the only important varieties appearing in the urine. *Fat* may be an accidental contamination or may occur pathologically in lipuria and chyluria. Under the microscope fat appears as highly refractile droplets with sharply defined dark margins. It is stained black by 1-per-cent. osmic acid, and bright red by a saturated alcoholic solution of Sudan III, and is soluble in ether or chloroform. Occasionally *fatty-acid crystals* are seen as straight or wavy needles which often have a stellate arrangement. *Contamination* of the urine with dust, faeces, dusting powders, or sputum may introduce a variety of objects such as vegetable tissue, muscle-fibres, or starch.

For details of the less usual tests a textbook of Pathological Chemistry should be consulted.

P. J. CAMMIDGE.

**URINE, EXTRAVASATION OF.**—This condition may follow injury or disease of any part of the urinary tract, but the most important site is the urethra. Traumatic rupture of the membranous urethra may accompany fracture of the pubic portion of the pelvis, whilst severe violence applied to the perineum may result in a laceration of the bulbous or penile urethra. The rupture may only affect a small portion of the circumference of the canal, and the leakage, at first, may be slight and pass unnoticed because of the bruising which occurs. Rough instrumentation, either by metal or gum-elastic catheters or bougies, may cause a breach of the mucous membrane and consequent extravasation.

When the result of disease, extravasation occurs where the wall is weakened by ulceration, and generally the ulcer is immediately proximal to an organic stricture of the bulbous part of the penile urethra.

A periurethral abscess due to slight extravasation is often a precursor of the more serious escape which develops as the result of some extra straining effort. (See ABSCESS, PERIURETHRAL.)

The course of the extravasated urine is determined by anatomical limitations. When the leakage is from the membranous or bulbous urethra, the scrotum soon becomes enormously swollen and oedematous, and the urine

travels up on to the abdomen along each side of the base of the scrotum, the fascial attachments preventing it from extending to the thighs or towards the rectum. The subcutaneous tissues of the abdomen are infiltrated. In the worst cases the urine may travel almost to the axillæ, and occasionally the upper parts of the thighs are affected.

The infiltrated parts are swollen and puffy, but pain is not a prominent feature. Soon, especially if the urine be septic, a severe cellulitis is set up, and the scrotum sometimes becomes gangrenous. The patient is rapidly poisoned by the microbial toxins generated in the tissues. If the condition be not treated promptly, death is likely to occur from toxæmia, or later from pyæmia.

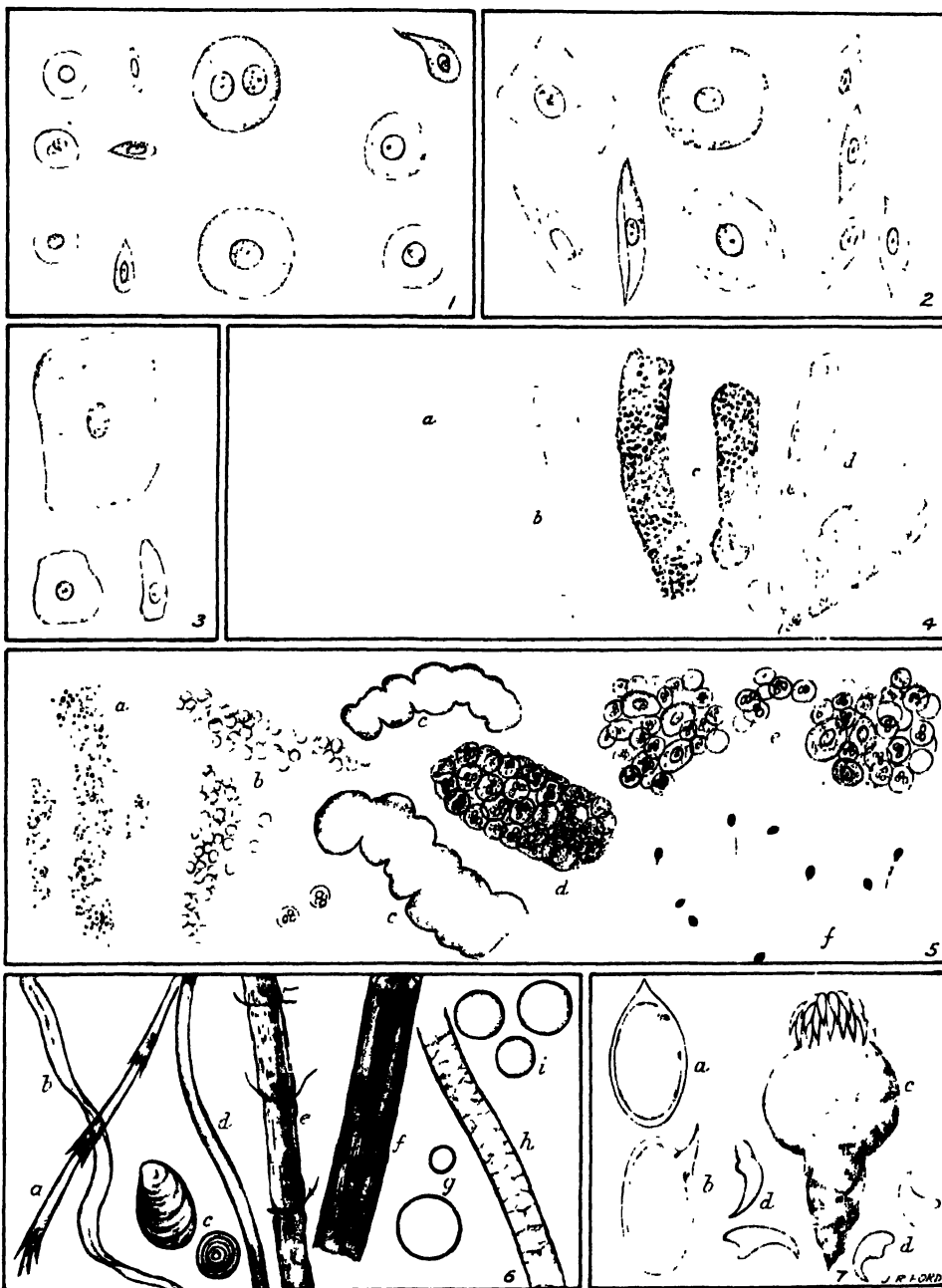
**Diagnosis.**—In every case of fractured pelvis or severe blow in the perineum the possibility of ruptured urethra must be considered. In every case of stricture one should be on the look-out for possible extravasation. A periurethral abscess must be regarded as a first stage which will lead on to the more serious condition if untreated; such an abscess at first lies deeply, may not give rise to fluctuation, and often feels like a solid tumour. In severe cases of extravasation the swollen scrotum and abdominal wall, with the absence or diminution of urine passed per urethram, sufficiently indicate the disease.

**Treatment.**—Every periurethral abscess must be treated by early and thorough incision. The immediate indication in all cases of extravasation is to let the urine out of the cellular tissues, and free incisions must therefore be made into the infiltrated parts. Four or five deep incisions into the swollen scrotum are generally needed. The second indication is to drain the bladder by the insertion of a perineal catheter, or if possible by tying in an ordinary catheter after exposing and dealing with the diseased portion of the urethra by perineal section. If skilled surgical help is not available the free multiple incisions must be made and the cause of the condition treated later.

After-treatment consists in giving frequent mild antiseptic hip-baths, administering nourishing and easily digestible food, prescribing tonics such as quinine and strychnine, and treating any infection by injections of the requisite vaccine.

ZACHARY COPE.

**URINE, INCONTINENCE OF** (see INCONTINENCE OF URINE).



1, Cells from kidney and ureter. 2, Cells from bladder. 3, Cells from vagina. 4, (a) Hyaline casts; (b) finely-granular casts; (c) coarsely-granular casts; (d) epithelial casts. 5, (a) Pseudo-casts (amorphous urates or phosphates); (b) blood casts; (c) waxy casts; (d) pus casts; (e) "clap" thread, or urethral shred; (f) spermatozoa. 6, (a) Feather; (b) cotton; (c) starch; (d) silk; (e) hemp; (f) hair; (g) air-bubbles; (h) wool; (i) fat. 7, (a) Ovum of *Schistosoma haematobium*; (b) ovum of *S. mansoni*; (c) scolex of *Taenia echinococcus*; (d) detached hooklets of *Taenia echinococcus*. ( $\frac{1}{2}$  objective.)



## URINE; VARIATIONS IN AMOUNT OF

**URINE, RETENTION OF** (*see* RETENTION OF URINE).

**URINE, SUPPRESSION OF** (*see* URINE, VARIATIONS IN AMOUNT OF).

**URINE, VARIATIONS IN AMOUNT OF.**—In healthy adults each kidney secretes about 0.5 c.c. of urine per minute, so that the two kidneys secrete 60 c.c. (2 oz.) per hour. The urine flows into the bladder in gushes at intervals of 10-30 seconds, and the bladder is emptied on the average five times in the twenty-four hours. The average daily volume of urine excreted by a healthy adult male varies between 1,000 and 1,500 c.c. (35-50 oz.). Women usually pass less, 900 to 1,200 c.c. (30-40 oz.). Abnormally small individuals and children excrete correspondingly smaller volumes. Taking the average weight of a healthy adult as 75 kilos (165 lb.) and the average excretion as 1,200 c.c., the normal amount, X, for a person of weight W, is given by the formula— $X : 1200 :: W : 75$ . Children, especially nursing infants, pass proportionately larger amounts owing to the preponderance of liquids in their diet. Some 67 per cent. of new-born infants pass urine the first day of life, but generally only after the lapse of twelve hours or more. The remaining 33 per cent. do not urinate until the beginning of the second or third day. The average quantities passed for different ages are : 1st day, 12 c.c. ; 3rd day, 23 c.c. ; 5th day, 35 c.c. ; 7th day, 51 c.c. ; 10th day, 61 c.c. ; 5th month, 100 c.c. ; 1 year, 237-266 c.c. ; 3 years, 355-414 c.c. ; 5 years, 414-469 c.c. ; 7 years, 499-552 c.c. ; 10 years, 688-746 c.c. ; 12 years, 861-890 c.c.

The passage of a large excess of urine in the 24 hours is termed *polyuria*. When the urine is of low specific gravity it is known as *hydruria*. By *oliguria* is meant the passage of too little urine. *Anuria* denotes the passage of no urine.

*Suppression* of urine is due to failure of the renal functions, and may be either obstructive or non-obstructive. The former occurs in conditions in which the ureters are involved, and any urine passed is likely to be pale and watery with no abnormal constituents. In non-obstructive suppression the urine voided is likely to be concentrated, high-coloured, and to contain abnormal constituents such as casts, or blood.

*Retention* of urine (q.v.) is due to inability of the bladder to empty itself, and may give rise to anuria if unrelieved.

**Alterations in amount.** (A) *Physiological.*

—The amount of urine passed is *increased* physiologically in low altitudes, by hearty eating, copious drinking, moderate exercise, intellectual work, diuretics, inhalation of oxygen, and electrical stimulation. It is *decreased* in high altitudes, by limiting the intake of fluid, abstinence from food, profuse perspiration, and excessive exercise.

The excretion varies inversely to the insensible perspiration, being increased in cold and diminished in hot weather. As a rule, during the night only a quarter to a half as much urine is passed as during the day. This ratio is generally reversed in diseases of the heart and kidneys, in aged persons with arteriosclerosis, in cachexias, and in diabetes insipidus. In these conditions the amount of urine passed in the night may be double that excreted in the day.

(B) *Pathological.*—The quantity of urine passed in pathological conditions depends upon (1) the condition of the renal parenchyma and (2) the velocity of the blood-current in the kidneys; it is therefore affected by general circulatory disturbances as well as by disease of the kidneys. An increase in the blood-pressure causes a corresponding rise in the quantity of urine passed if the blood-flow through the kidneys is not interfered with. Diseases of the heart and lungs leading to passive congestion are associated with a diminished excretion of urine, owing to slowing of the renal circulation. Renal diseases do not affect the volume of the urine appreciably unless both kidneys are involved, the healthy organ assuming vicariously the total function. In inflammatory affections of the kidneys, the more acute the nephritis the more the amount of urine falls below normal, and the more chronic the course of the inflammatory changes the more the amount exceeds the normal. The quantity excreted in cases of contracted kidney may be as high as 12 litres (approximately 420 oz.) a day.

The excretion of urine is *temporarily diminished* after anaesthetics, in febrile diseases, in conditions associated with diarrhoea and vomiting, during convulsions, colic, hæmorrhage, shock, and collapse. A *persistent decrease* is met with in most chronic diseases, in lead poisoning, in melancholia, and generally in hepatic diseases, especially catarrhal jaundice and chronic affections of the liver. *Suppression* of urine occurs in uræmic coma, in poisoning by corrosive sublimate, in cholera and in acute peritonitis.

## URTICARIA

A temporarily increased excretion of urine is seen in hysteria, nervous excitement and migraine; after epileptic attacks; at the commencement of convalescence from typhoid fever, pneumonia and catarrhal jaundice, and after the reduction of dropsy. The volume of urine is *intermittently increased* in hydro-nephrosis. A marked *permanent increase* in the quantity of urine is characteristic of diabetes insipidus. An increase, varying as a rule with the severity of the case, is seen in diabetes mellitus; but a normal, or even a subnormal, amount may be passed, even when a considerable amount of sugar is present. Such cases are referred to as "diabetes decipiens." Generally, the amount of urine passed rises with an increase in the output of sugar, but at a slower rate.

*Abnormal frequency* of urination may occur with or without polyuria. The former is seen in nervous conditions, fright, worry, diabetes, and chronic interstitial nephritis. The latter is met with in spinal diseases affecting the nerve supply of the bladder, in congestive and inflammatory affections of the urinary tract and adjoining structures, and during renal colic. It may arise from the irritation due to calculi, hyperacid urine and urethral conditions, and in malignant or tuberculous disease of the genito-urinary tract. Frequency of micturition with pain occurs in diseases of the renal pelvis or lower urinary tract, rarely with diseases of the renal parenchyma.

P. J. CAMMIDGE.

**UROBILINURIA** (*see URINE, EXAMINATION OF*).

**URTICARIA.**—A name conveniently applied to an itchy eruption consisting predominantly of wheals or "hives" of the type produced by the sting of a nettle, from which the name is in fact derived. The eruption may result from contact with external irritants, or from internal causes, and should be regarded as a symptom rather than a disease. In very young children the wheal is uncommon, being replaced by a papulovesicular lesion which is even more itchy than the wheal.

The *wheal* at its full development is a firm white swelling of variable dimensions surrounded by a pink areola of irregular shape; ordinarily the wheal is of moderate size, averaging  $\frac{1}{2}$ – $\frac{1}{4}$  in. in diameter, but in exceptional cases, constituting a distinct clinical group,

"giant urticaria," it may be a raised plaque several inches wide. The duration of individual wheals is always transient, but there may be recurrences so frequently repeated as to give the impression of persistence (*urticaria perstans*).

The wheal is produced by a circumscribed inflammatory oedema of the cutis, the epidermis usually remaining unaffected. When the tension is extreme the basal layers of the epidermis are ruptured and vesication results.

The *distribution* of the lesions varies with the cause, localized eruptions being the commoner in cases due to external irritants, more generalized eruptions in cases ascribed to internal causation. Thus the distribution may be very extensive or very restricted. There are no consistent sites of election, though it may perhaps be said that the trunk is more copiously covered in adults, the limbs in children. Authorities differ as to the propriety of including the clinical type known as angioneurotic oedema (*see OEDEMA*) under the category of urticaria. If exclusion be made (as I think it should be) of this group, urticaria does not often affect mucous membranes. When the inflammatory oedema occurs in sites where the tissues are lax, as around the orbit, the lips, the vulva, and the prepuce, great distension may take place.

**Symptomatology.**—The constitutional symptoms caused by the eruption are mainly summed up in the effects of the intense pruritus; secondary pus-infections of the skin are common, especially in children, in whom scratching is more uncontrolled than in adults. In the latter, however, sleeplessness and the fear of sleeplessness may exert a more profound disturbance. In acute attacks, rise of temperature has been described, but must be very uncommon; I have never met with it. The eruption usually disappears without leaving any trace, but when recurrences have been frequent, and the skin has been much scratched, a secondary pigmentation is sometimes found, which is to be carefully distinguished from urticaria pigmentosa (q.v.). In neglected children who have suffered deep-seated pus-infections, scarring is not infrequent.

**Etiology.**—Two sharply differentiated groups may be described, according as an external or an internal irritant is at work. In both groups the personal factor, the patient, plays an important part, individual idiosyncrasies being especially notable in determining the degree of response to the stimulus. "One man's meat is another man's poison." When

## URTICARIA

the irritant, whether external or internal, is continually renewed the rash may become chronic, a result that is commoner with the internal causes, which are usually more difficult to determine, and therefore more difficult to avoid. It would be impossible to compile a complete list of such causes, which is constantly being enlarged.

In the first group may be enumerated contact with a large variety of plants and minerals; the bites of insects; mechanical friction and injury. In the second group, food and drink and drugs play by far the most important part. It is probable that a common factor in many of these causes is a hyperacidity such as is produced by acid wines, fruits, and the products of disordered metabolism. It is commonly assumed that the result is a destruction or elimination of calcium, the "decalcification urticaria" of Wright. In many of these cases, but by no means consistently, there is a diminished coagulability of the blood. Personally, I am of the opinion that there is another and smaller group of cases in which a pronounced hypoacidity exists, as revealed by the Joulie tests. A small minority of cases, perhaps of increasing frequency in these days of indiscriminate vaccine-therapy, is due to organic poisons, with which may be classed some very remarkable cases of midge-bite eruptions with severe constitutional symptoms, seen in England in the summer of 1920.

**Diagnosis.**—*Scabies*, especially in children, may be very difficult to distinguish from the vesicular urticaria which, as has been stated, is the common type in infancy. In infantile scabies, if the diagnostic burrow is not demonstrable, the distribution on the wrists, and especially about the ankles, is very suggestive of scabies. The absence of infection is a contraindication of scabies. From *dermatitis herpetiformis*, urticaria is distinguished by the comparative paucity of wheals and by the mixture of lesions so characteristic of dermatitis herpetiformis, which will show simultaneously papules, wheals, and vesicles, the latter being commonly arranged with some attempt at herpetiform grouping. If the vesicles attain any considerable size the diagnosis of dermatitis herpetiformis is more probable. The itching in the latter disease is even more terrible than in urticaria. Some constitutional diseases which are accompanied by intense pruritus may be confounded with urticaria, e.g. early stages of *mycosis fungoides*, *leukæmic prurigo*, and *prurigo of Hebra*. But these are all rare dis-

## URTICARIA PIGMENTOSA

eases, and accompanying symptoms are in most cases present to differentiate them.

**Dermographism.**—In many persons there is a peculiar liability to wheal-formation upon moderate friction of the skin, a symptom which has been sometimes called "factitious urticaria." This has probably no relations with urticaria, and, as Darier points out, the artificial wheal is not pruritic. The significance of the symptom is not at present very well understood.

**Treatment.**—From what has been said it will readily be inferred that it is of the first importance to identify the cause, if possible, when its removal will be the obvious step to take. If this is difficult to establish, one is obliged to resort to symptomatic treatment, and an antacid mixture combined with a vigorous purge is indicated. When this experimental treatment has not been successful it is, in my opinion, worth while to undertake Joulie estimations of acidity, and to be guided by the result as to the form of medication and the diet to adopt. I have often succeeded with acid mixtures (phosphoric acid in large doses, 30-60 min. of acid. phos. dil., with water, thrice a day), in cases showing Joulie hypoacidity. Calcium lactate is in many hyperacid cases almost a specific, and should be given in full doses, either 1 dr. every two days or 15-20 gr. thrice a day. Magnesium hydrate tablets, as recommended by Joulie, are very useful in mild hyperacidity, 15 gr., three times a day, being an average dose. Locally, weak tar lotions (liq. carb. deterg., 15 min. to the ounce), with or without alkaline additions, are probably the most effective. Ointments are as a rule not well borne, but I would make exception of a cream (crude coal tar 1-3 dr., zinci oxidi 6 dr., lanolin and vaselin 1 oz. of each) which I have used with satisfactory results, especially on chronic much-scratched patches. In the management of urticarial children and sensitive adults it is especially important to prevent scratching; the prohibition of wool worn next to the skin, and the avoidance of overheated rooms and superfluous bedclothing, will do much to attain this end.

E. GRAHAM LITTLE.

## URTICARIA PERSTANS (see URTICARIA).

**URTICARIA PIGMENTOSA.**—A very rare disease, which belongs rather to nævoid conditions than to the urticaria group, the name being a misnomer. It is characterized by the usually slow development in early



## UTERUS, ELONGATION OF CERVIX OF

childhood of an eruption of macules or nodules of a prevailing buff colour which may deepen to a walnut brown, distributed chiefly upon the trunk, remaining persistent for indefinite periods (contrasting thus with the true urticarial wheal), seldom itchy to any great degree, and without any prejudicial influence on the general health. In a small minority of cases the eruption has been noted at birth, and, again in a small minority of cases, as commencing after adult age.

The **diagnosis** is hardly to be regarded as established without histological examination, which reveals an enormous infiltration of the corium with mast cells, and a deposit of pigment in the basal layers of the epidermis and sometimes free in the corium. Although the lesion is only moderately itchy, friction of a macule or nodule usually results in the speedy turgescence and reddening, transient in duration, of the lesion, and this clinical test is much relied upon in the diagnosis. Recently a group of cases has been described in which mast cells have not been a conspicuous feature in sections, and diagnosis might be extremely difficult in such cases. The macular eruptions are easily confounded with the *macular syphilide*, from which, however, they are readily distinguished by their duration and by their usual incidence in childhood. The nodular type of urticaria pigmentosa is often indistinguishable by clinical data alone from *xanthoma tuberosum* (see **XANTHELASMA**), from which the nodules are to be differentiated by the characteristic histology of the two conditions.

No treatment is of the slightest use.

E. GRAHAM LITTLE.

**UTERINE FIBROIDS** (see **UTERUS**, **NEW GROWTHS OF**).

**UTERUS AND VAGINA, MALFORMATIONS OF** (see **AMENORRHOEA**).

**UTERUS, CARCINOMA OF** (see **UTERUS**, **NEW GROWTHS OF**).

**UTERUS, DISPLACEMENTS OF** (see **PELVIC ORGANS, FEMALE, DISPLACEMENTS OF**).

**UTERUS, ELONGATION OF CERVIX OF.**—The cervix uteri is liable to elongation of its vaginal and supravaginal portions.

**ELONGATION OF THE VAGINAL PORTION.**—This is almost always a congenital deformity. From being scarcely recognizable it may be

so marked as to cause presentation of the external os at the vulva, when this part of the cervix will measure as much as 4 in. It is usually not seen by the practitioner until the patient marries, when she may seek advice on account of painful or difficult coitus, or later because of sterility, of which this condition is a cause. Occasionally it is discovered when making an examination for dysmenorrhœa.

The **diagnosis** has to be made from prolapse of the uterus, of the utero-vaginal type, in which the uterus first prolapses into the vagina, on account of the low position of the external os. It is easily established as hypertrophic elongation by noticing firstly that the vault of the vagina is the proper distance from the vulva, and secondly that the cervix cannot be pushed up, as would be possible if its low position were due to a prolapse into the vagina. Moreover, bimanual examination reveals the body of the uterus in its normal position.

**Treatment.**—The condition calls for treatment chiefly for sterility, but small degrees of elongation in an unmarried woman, causing no symptoms, should be left alone. If there are symptoms referable to the abnormal length of the cervix, it should be amputated at the level of the vaginal insertion.

**ELONGATION OF THE SUPRAVAGINAL PORTION.**—This is usually a secondary result of prolapse of the vagina in cases where at first the uterus is fairly firmly fixed in its normal position. The mechanism has been closely debated, but it is now thought to be due to a passive stretching of that portion of the cervix where it is held up by the transverse cervical ligaments and that portion which is attached to the prolapsing vagina. The dragging downwards of the vagina thus gradually pulls out the supravaginal portion. Evidences of this are (1) that the elongated cervix is much thinner than normal, and (2) that after the cure of the prolapse by operation a considerable contraction can follow.

The **treatment** is part of that for the causal condition, prolapse. Colpoperineorrhaphy, and often ventrosuspension (in cases of retroversion) are required, but in view of the probable correctness of the theory of stretching, it is not usually necessary to amputate the cervix, unless the elongation is very pronounced.

A. W. BOURNE.

**UTERUS, FIBROIDS OF** (see **UTERUS**, **NEW GROWTHS OF**).

## UTERUS, NEW GROWTHS OF

**UTERUS, INVERSION OF** (*see* PELVIC ORGANS, FEMALE, DISPLACEMENTS OF).

**UTERUS, NEW GROWTHS OF.**—Uterine neoplasms may be classified thus:—

**SIMPLE.**

1. Fibroids (Fibromyomata).
2. Adenomyoma.

**MALIGNANT.**

3. Sarcoma of body.
4. Carcinoma of body.
5. Carcinoma of cervix.
6. Sarcoma of cervix.

### 1. FIBROIDS

**Etiology.**—The etiology of these tumours is not known. They are commonest between the ages of 25 and 45. It is stated that fibroids cause sterility, but it is difficult to obtain sound evidence of this. There is evidence, on the other hand, that sterility causes fibroids. They are commoner in women who have not borne children. It seems probable that there is some common factor, as yet unknown, which predisposes the patient towards sterility and also to the formation of fibroids.

**Pathology.**—Sections of these tumours show whorls of spindle-shaped cells with a small nucleus. The cells are not striated. There is a very small amount of intracellular tissue, but a fair number of blood-vessels, and the general appearance is that of uterine muscle. This picture is best seen in "young" fibroids. When the tumour has existed for a considerable time, the appearance is altered by degenerative changes; the actual degenerations are described in detail below.

*Clinically*, fibroids may be—

- Subserous, or subperitoneal.
- Interstitial.
- Submucous.

A combination of any two or all of these varieties.

They are usually multiple, although one may be so much larger than the others as to give the appearance of a single tumour. Fibroids are also differentiated into two classes according to whether they occur in the body of the uterus or in the cervix.

#### CORPOREAL FIBROIDS

**Symptomatology.**—**Subperitoneal fibroids** usually give rise to symptoms solely from their size and weight. The patient may complain of "indigestion," or of increasing sluggishness of the bowels, not amounting to constipation; at the same time there may be a feeling of

weight, as though the rectum had not been completely emptied. A history of frequency of micturition can often be obtained. Attacks may come on at intervals and last for two or three days; they are probably due to the tumour intermittently falling forwards and pressing on the bladder.

With large tumours, a feeling of weight in the lower abdomen is usually complained of. Pain is provoked when there is a tumour impacted in the pelvis, and is then located in the region of the sacrum. A heavy dull pain occurs during menstruation, due to the congestion of all the pelvic organs, in which the fibroid shares, and very often the passage of clots is accompanied by sharp "contraction" pains.

Severe pain may be present when the tumour has a long pedicle, which has become twisted, causing strangulation of the vessels, or when suppuration occurs in the substance of the tumour. Either of these conditions usually comes on more or less acutely; indeed, suppuration is often secondary to torsion. The violence of the pain may subside in a degree, but a dull aching pain is left due to the inflammatory reaction spreading to the neighbouring viscera, an extension which may terminate in a general peritonitis.

Acute and complete torsion of the pedicle of a fibroid is one of the abdominal catastrophes. Partial torsion of the pedicle is more common and is associated with pain due to the inflammatory reaction which follows, giving rise to adhesions.

Sessile subperitoneal fibroids, when they attain large size, sometimes undergo axial rotation, and in the course of this manœuvre twist the body of the uterus on itself. This accident, however, is rare. A fibroid which has undergone rotation may present a very pale, almost pearly appearance, with flakes of coagulated exudate on its surface. On other occasions it may be dark purple, with veins like straps coursing over its surface. This occurs when the twist has been sufficient to occlude the veins, but not to occlude the arteries. Later, adhesions may form to the surrounding viscera, especially to the great omentum. The stalk, be it pedicle or uterus, is swollen and oedematous. The tumour is liable to infection by migration from the gut, in which case a serious catastrophe can only be averted by operative interference.

The degenerations described below (p. 464) are accompanied by pain, especially red de-

## UTERUS, NEW GROWTHS OF

generation. Calcified fibroids are frequently painful and tender because of the peritoneal irritation they cause. Calcification usually takes place in pedunculated fibroids.

**Interstitial fibroids** give rise to the same symptoms as subperitoneal fibroids, in so far as these are due to the mechanical effects of the size of the growths. They also lead to excessive loss at the menstrual periods, and frequently to a shortening of the menstrual cycle and delay in the onset of the climacteric. Thus a patient may give the history that her periods used to occur at twenty-eight days' intervals and that the loss lasted for four or five days, but that, lately, the periods have been occurring at shorter intervals—say every three weeks, and that the loss has lasted for eight or nine days and been more profuse. At the same time there is often dysmenorrhœa, due to the clotting of the "loss" and to the forcing of the clots through the cervix. The increased loss is explained by the greater size of the cavity of the uterus and by changes which occur in the endometrium (*see below*), and also probably by a mechanical effect—the tumour preventing the normal contractions of the uterine muscle from taking place. The shortening of the cycle can only be explained by congestion of all the pelvic organs, due to the increased blood supply called forth by the requirements of the tumour. Although fibroids themselves are not very vascular tumours, there is a very rich blood supply to the capsule, and it is usual to find an artery larger than the normal uterine artery running up to the tumour and a large plexus of dilated veins draining the blood away. It is obvious that all the vessels supplying the uterus, including the ovarian arteries, must be affected by this call for a more liberal blood supply, and the increased vascularity of the ovaries would naturally stimulate them and so shorten the menstrual cycle.

A **submucous fibroid** calls attention to its existence by bleeding before it becomes large enough to produce mechanical effects; one no larger than a cherry may produce very severe menorrhagia.

Another train of symptoms is caused by the uterus regarding it as a foreign body, and increasing its periodic contractions in an effort to expel it.

During normal menstruation the uterus contracts to expel the menstrual flow and also to close the vessels and prevent undue loss of blood. When a foreign body is present these contractions become much more forcible, and

therefore painful, and dysmenorrhœa accompanied by increased loss, coming on in the early thirties, should always suggest a small submucous fibroid.

As this process continues, the fibroid gradually becomes squeezed by the contractions of the uterus, until it is no longer sessile, but becomes stalked, a condition arising which is dealt with under **UTERUS, POLYPI OF**.

When a larger fibroid, attached to the uterus by a broad firm base, comes to project into the uterine cavity, it is not infrequently gripped by the uterus in an attempt to extrude it. Such attempts may not be successful in drawing out a thin pedicle, or stalk, but may have the effect of dragging down the fundus with the fibroid and so inverting the uterus. This is fraught with very serious risks for the patient, as the tumour is very liable to septic infection, which converts the whole mass into a slough, with all the associated symptoms of septic absorption, and increases the risk of operative measures.

*Changes in the endometrium occurring in association with interstitial or submucous fibroids.*—The endometrium covering a fibroid, whether submucous or interstitial, undergoes proliferation and increased vascularization. Although fibroids themselves are not highly vascular tumours, their capsule is always liberally supplied with large blood-vessels, and it is probable that this increased vascularity of the capsule is one of the primary causes of the changes in the endometrium. The other factor is œdema, due partly to pressure. In the normal uterus the endometrium measures 1.5–2 mm. in thickness; that covering a fibroid may be as much as a centimetre.

Microscopically, the epithelium remains much the same. The surface cells retain their cilia, but the mouths of the glands are more widely separated and are slightly dilated. In the deeper layers the glands themselves are seen to be enlarged and proliferated.

### FIBROIDS OF THE UTERINE LIGAMENTS

Fibroid tumours also grow in the muscle contained in the uterine ligaments, the chief of which are the round ligaments, the broad ligaments, and the cervico-pelvic ligaments. They are indistinguishable from subperitoneal fibroids until exposed at operation.

### CERVICAL FIBROIDS

By a distinction which is clinical and not pathological, a fibroid growing in that portion

## UTERUS, NEW GROWTHS OF

of the uterus below the level of the internal os is known as a "cervical fibroid." The etiology and pathology are identical with those of fibroids of the body.

Cervical fibroids may be single or multiple and may or may not be accompanied by fibroids of the body; in the majority of cases they are single.

Spherical in shape at first, as they grow they reach the sides of the bony pelvis, which restrict their expansion laterally so that they tend to become elongated or oval. As growth proceeds further they mould themselves to the shape of the pelvis, and occasionally have a shallow depressed groove on the posterior surface where they are in contact with the rectum.

Like fibroids of the body, they may be submucous, intramural, or subperitoneal. They may be situated anteriorly or posteriorly or laterally. When anterior they may push their way upwards between the uterus and the bladder and so come to lie outside the pelvis altogether, in which case they retain their spherical shape. Very rarely they have been removed from this situation without opening the peritoneal cavity. When a fibroid grows in the other situations, the cervix tends to be elongated and stretched out over the surface of the tumour, while the body of the uterus is pushed up into the abdomen, and is perched on top of it. By abdominal palpation the corpus uteri may be felt as a knob on the summit of the tumour.

**Symptomatology.**—The main symptoms are increase in the menstrual loss and a feeling of discomfort in the lower abdomen. The menorrhagia is not so great, and, indeed, may be absent, as the tumour may grow to a large size without materially increasing the area from which the menstrual flow arises.

Perhaps the most typical symptoms of these tumours are their effects upon micturition. The bladder is drawn up into the abdomen as the tumour rises, and the urethra becomes lengthened, so that the bladder may become an entirely abdominal organ. At the same time it is stretched out laterally, especially at its base, and its contractions are mechanically interfered with.

When the tumour grows sufficiently to fill the pelvis it becomes impacted between the promontory of the sacrum and the symphysis pubis and, by causing direct pressure upon the urethra, leads to increasing difficulty in micturition, which, if not relieved, will go on

to complete obstruction of the urethra and retention of urine. In such a condition the utmost care must be exercised in passing a catheter. One made of rubber is preferable, for a glass one may easily be broken by the pressure of the tumour, the broken end being left in the bladder.

Chronic constipation is a frequent accompaniment of cervical fibroids, and is due to the pressure on the rectum. Complete intestinal obstruction does not occur, as there is more room at the sides of the promontory and in the hollow of the sacrum.

On abdominal palpation the tumour can usually be felt low down, but a cervical fibroid as big as an orange in the posterior wall of the cervix may be completely within the cavity of the pelvis and may escape detection abdominally.

Vaginally the conditions differ with the various situations in which the tumour may arise. Submucous cervical fibroids grow into the lumen of the cervical canal and expand the cervix uniformly. On examination, the vault of the vagina is found to be occupied by a rounded tumour. There is no nipple-like projection of the cervix, but the external os is recognized as a dimple on the surface, and around it are thin edges representing the expanded cervical walls.

The size of the os varies; in most cases it is small, but if the tumour arises very low down, or, if it has commenced to become polypoid, the os may be dilated to quite a considerable degree.

When dilated it becomes crescentic in outline and thus provides a feature by which this condition can be distinguished from a fibroid polyp. On examining a fibroid polyp the thin edge of the os can be felt completely encircling the tumour, and a sound, or probe, can be passed all round the tumour. With a submucous cervical fibroid the probe can only be passed part of the way round, as the base of the tumour is attached to the cervical canal at one side.

Cervical fibroids growing on the anterior surface of the cervix may not project into the vagina at all, but may grow upwards between the uterus and the bladder and rise above the brim of the pelvis. The cervix, which is normal in shape, is then found to be pushed backwards, and the rounded tumour can be easily felt bimanually in front of the uterus.

Interstitial fibroids arising in the portio

## UTERUS, NEW GROWTHS OF

vaginalis of the cervix project into the vagina below the cervix. They appear very like a polyp on a thick stalk. On careful examination, however, the stalk can be distinguished as being the cervix itself, and as not coming through the cervix. The os can be felt on one side of the "stalk," while on the other side the stalk is continuous with the vault of the vagina; these signs, taken together, make it clear that the tumour is growing in the cervix and not projecting through it.

Such a tumour may become infected with septic organisms; the symptoms are identical with those of a sloughing fibroid polyp (*see* p. 477).

Fibroids growing in the walls of the supravaginal portion of the cervix at the sides or posteriorly distort the uterus more than any other variety. The cervical canal, normally about 1 in. long, may be stretched out over the tumour to a length of 6 or 8 in. The vaginal portion of the cervix is shortened and partially taken up in the process and appears as a dimple with slightly raised edges. Immediately above this is the globular mass of the tumour, occupying the whole vault of the vagina.

This condition of the cervix and os is a very useful point in diagnosis. It distinguishes a cervical fibroid from a subperitoneal fibroid impacted in the pouch of Douglas, for though in the latter the vault of the vagina is also filled with a globular tumour, the cervix is drawn upwards and forwards and comes to lie behind the symphysis pubis.

The dimple-like os is diagnostic of a cervical fibroid.

Subperitoneal fibroids growing from the posterior wall of the cervix may attain a considerable size without distorting the cervix. They extend into the pouch of Douglas and fill the cavity of the pelvis. The cervix is pushed forward towards the symphysis pubis.

It is not always possible to distinguish these tumours from subperitoneal corporeal fibroids in the pouch of Douglas. Indeed, as the posterior surface of the cervix is continuous peritoneally with the posterior surface of the body of the uterus, the actual distinction between the two is arbitrary.

### DEGENERATIONS OF FIBROIDS

**Red degeneration, or necrobiosis,** as it is called, is a degeneration peculiar to these tumours. Though usually associated with pregnancy, it may occur apart from it.

A softening of the tumour takes place and is accompanied by a change of colour from pale yellowish-pink to a deep purplish-red. This colour change commences in patches and spreads rapidly through the whole tumour; it is due to a breaking down of blood-cells together with a lysis of the cells of the tissue proper of the tumour and a diffusion of blood pigment throughout the softened mass.

The etiology of this change is still obscure. Investigation shows that in some cases micro-organisms exist, but in many they do not. It is now generally accepted that the micro-organisms are secondary to the change and not the cause of it. It is important to remember the ease with which micro-organisms invade these tumours.

Red degeneration may take place either during pregnancy or during the puerperium, and one or more of the tumours present may or may not be affected by this peculiar change. It is usually accompanied by a rise of temperature, especially during the puerperium. The fibroid or fibroids affected are painful and are extremely tender to the touch.

**Calcification** not infrequently occurs in subperitoneal fibromyomata after the menopause; the longer they remain—i.e. the older the patient—the more likely is this to happen. It has been looked upon as Nature's cure for the tumour, but this theory is not a good one, as most of the symptoms remain, and other possible accidents may take place, due to the altered consistency of the tumour.

The deposit of lime salts takes place gradually and progresses, so that in time the whole tumour may be converted into a solid mass of lime salts—the "womb stones" of the old writers. Specimens exist in most museums of fibromyomata which have become calcified, and in which all the soft tissue has been absorbed so that a delicate framework of chalky material is left.

Obviously, such a tumour will produce all the symptoms of its softer precursor aggravated by the hardness of the growth. Calcified fibroids frequently give rise to a good deal of pain and are tender to the touch.

At the same time, the patient has to run certain additional risks. Cases have been reported in which an elderly woman has had a fall which, although not very violent, has produced symptoms of the greatest severity—so severe, in fact, as to warrant immediate operative interference. At operation, or autopsy, it has been discovered that the bowel has been

## UTERUS, NEW GROWTHS OF

ruptured by being caught between the calcified fibroid and the anterior abdominal wall.

Large veins on the surface of the tumour may be ruptured in a similar way, causing severe internal hæmorrhage.

**Mucoid degeneration.**—In this condition the tumour contains areas of varying size, which present the appearance of glistening transparent islets of mucus. The islets may be quite small or may be so large as to occupy the whole of the tumour. The material is semi-solid and consists of liquefied debris of the original spindle-shaped cells. The degree to which this degeneration progresses varies from a few islets the size of a pea or less to the conversion of the whole tumour into a thin-walled sac containing gelatinous fluid. To these advanced cases the term "cystic degeneration" is often applied.

Mucoid degeneration sometimes assumes a more generalized form. The fibres themselves undergo a degeneration, the cell-protoplasm becoming cloudy and the outline of the nucleus indistinct. The connective-tissue cells are increased in amount, and more fibrous tissue is formed.

The whole tumour is affected by this change and is *œdematous*.

**Hyalin degeneration.**—The cut surface of the tumour presents a grey or greyish-pink appearance and the tumour is avascular but firm. The cells retain their outline to some extent, are clear, and do not readily take up the usual stains.

**Nævoid degeneration.**—A portion or the whole of a fibroid is sometimes found to have become a soft, friable, dark purple mass, somewhat resembling a placental site. The capsule of the tumour is full of large blood sinuses.

### FIBROIDS COMPLICATING PREGNANCY AND LABOUR

Although fibroids are usually associated with sterility, cases often occur in which they complicate pregnancy.

The special liability of fibroids associated with pregnancy to undergo red degeneration holds good for any fibroid, whether it be corporeal or cervical, submucous, interstitial, or subperitoneal. A fibroid so degenerated is prone to invasion by septic micro-organisms, which may migrate from the bowel, or may infect the tumour during or after parturition, whether the patient aborts during the early months or goes to term, and a most serious form of septicæmia may result.

Infection is, therefore, a definite danger to a woman who has become pregnant when she has fibroids, for though she may go to term without any pronounced symptoms, and labour may be got through without any mechanical obstruction, the risk is still present in the puerperium.

The extent to which fibroids affect pregnancy and labour varies between wide limits according to their number, size, and situation. One or two small fibroids situated in the upper pole of the uterus will cause no mechanical interference with pregnancy or labour, and provided the patient escapes some unusual complication, the pregnancy may terminate in the birth of a living full-term child, followed by a normal puerperium.

On the other hand, a mass of large fibroids occupying the substance of the uterus does not allow the uterus to expand to accommodate the growing fetus. When the limit of expansion is reached abortion takes place and brings with it great risk of post-partum hæmorrhage, as the uterus is unable to contract owing to the mechanical effect of the fibroids in its wall. The hæmorrhage may be very severe and is not easily controlled by the ordinary methods. Fibroids in these circumstances are particularly liable to septic infection of a virulent nature, which frequently ends fatally.

When the uterus contains fibroids of such a size that it can just be accommodated in the cavity of the pelvis, it is obvious that the rapid enlargement due to pregnancy will cause impaction under the promontory of the sacrum. When this occurs, the patient, who has missed two or three periods, is seized with a violent pain in the abdomen and is unable to pass urine. The pelvis is filled with a mass which is fixed. The full bladder obscures abdominal palpation, and the condition is easily confused with retroverted gravid uterus. The position and direction of the cervix may or may not serve to distinguish between them.

The greatest care must be exercised in passing a catheter, as not only does the tumour press firmly on the urethra, but it is also probable that the course of the urethra is distorted; it is not difficult to injure this structure, and thus further complicate an already complicated condition.

If the bladder be emptied and the case be one merely of retroverted gravid uterus, no tumour will be felt in the abdomen, but if the condition be due to incarceration of a

## UTERUS, NEW GROWTHS OF

pregnant fibroid uterus a tumour will remain palpable from the abdomen after the bladder has been emptied.

Attempts may be made, using the utmost gentleness, to push the tumour out of the pelvis; if this can be accomplished the retention will be relieved for the moment, but the right course in these cases is to operate.

A pedunculated subserous fibroid may cause no symptoms during pregnancy. If it becomes degenerated, or infected by septic organisms, it will give rise to pain, tenderness, and fever. The symptoms produced may indicate surgical interference, or may subside with rest and general treatment.

A fibroid of this type may call attention to its existence by getting into the pouch of Douglas below the pregnant uterus. During pregnancy it may give rise to pain or a feeling of weight or discomfort in the lower abdomen, and it can easily be recognized during the routine vaginal examination of the pregnant woman. A fibroid in this situation will offer an insuperable barrier to delivery of the child *per vias naturales*. When, therefore, the fibroid is not discovered until labour commences, operative interference is the only hope. Caesarean section, with myomectomy or hysterectomy, is called for.

A cervical fibroid will give rise to a train of events very similar to the pedunculated fibroid in the pouch of Douglas. Symptoms only appear during pregnancy if degeneration, infection, or impaction takes place.

Small cervical fibroids may soften sufficiently during pregnancy to permit delivery, but a cervical fibroid as large as a man's fist will offer an insuperable obstruction.

The great dangers with submucous fibroids and pregnancy are (1) that they are more liable than others to become the seat of degeneration; (2) that they may be the cause of severe post-partum hæmorrhage, as the uterus is prevented from contracting properly by the presence of the tumour in its cavity; and (3) that they are specially liable to become infected during the puerperium, and so give rise to a very severe form of sepsis.

### DIAGNOSIS OF FIBROIDS

The most characteristic symptom is menorrhagia. It is important to pay attention to this symptom, as the normal loss for individual patients differs. With fibroids we expect to find that the loss is greater than it used to be and is increasing. Not only is the

loss greater at the time, but the duration of the loss is longer.

As mentioned above, the cycle is shortened so that the menses, instead of commencing every twenty-eight days, come on at intervals of twenty-one or even fourteen days. In severe cases the lengthening of the duration and the shortening of the cycle may lead to almost continual loss.

Fibroids prolong menstrual life and delay the onset of the menopause. Instead of this coming on between the forty-second and forty-eighth year, it may be delayed until well into the fifties.

On abdominal examination a firm tumour is felt rising out of the pelvis. It is usually of irregular shape, being made up of a series of rounded tumours conglomerated together; but if there is a single large fibroid the outline may be quite smooth and regular.

Usually the mass has limited mobility. If very large and filling the pelvis, it may be so moulded to the pelvic cavity that it is immovable and gives the appearance of being fixed to the wall of the pelvis itself. On the other hand, a considerable degree of mobility is obtainable with a pedunculated subperitoneal fibroid with a long pedicle.

Fluctuation is only present on the rare occasions when cystic degeneration has proceeded to an advanced degree; as a rule there is no sense of fluctuation. The tumour does not give rise to pain and is not tender on palpation except when red degeneration or inflammatory changes in the peritoneum and surrounding viscera are in process.

Fibroids do not lead to the presence of free fluid in the peritoneal cavity. It is unusual to find intestine in front of them, especially when they are of large size; hence they are usually dull on percussion.

In a thin patient with a large fibroid the outline is frequently visible through the abdominal wall.

On vaginal examination the cervix is more or less pushed down into the pelvis; it is not infrequently asymmetrical, and may be pushed right to one side.

A mass of greater or less extent may be found filling up the posterior fornix. There is no bleeding on examination.

On bimanual examination the mass in the abdomen is found to be one with the mass in the pelvis. Movement of the abdominal mass either up and down, or from side to side, transmits the same movement to the cervix.

## UTERUS, NEW GROWTHS OF

In patients with a rigid abdominal wall it is frequently difficult definitely to move the abdominal tumour. When this is the case, by placing one finger in the vagina on the cervix, and with the other hand giving a series of short sharp impulses to the abdominal wall, commencing well above the umbilicus and gradually approaching the symphysis pubis, it will be found that, at a certain point, corresponding to the upper border of the tumour, the impulse is transmitted to the finger on the cervix. This transmitted impulse is of great value in difficult cases.

Further examination must be conducted with a view to ascertaining whether the body of the uterus can be made out separately from the rest of the tumour.

With the index finger of the right hand in the vagina the left hand explores the region of the lower abdomen just above the symphysis pubis. If anything corresponding to the size of the body of the normal uterus is felt, efforts should be made to move it separately and to ascertain if this movement is transmitted to the cervix. If the movement is not so transmitted it is probable that the tumour felt is a small sub-peritoneal fibroid on the anterior wall of the uterus.

In most cases the **differential diagnosis** presents no great difficulties. When we obtain the typical history of increasing loss at the periods and coupled with this there is a hard irregular swelling in the abdomen, a swelling which is continuous with the cervix, the diagnosis may be very easy.

It is the atypical cases which give rise to difficulty, and when such a case is complicated by a fully incorrect history on the part of the patient the difficulties may be increased.

Fibroids may have to be distinguished from —

- (1) Pregnancy, normal and abnormal.
- (2) Enlargements of the uterus due to other causes, e.g. malignant disease of the body, subinvolution, fibrosis uteri, pyometra, adenoma, and rare conditions such as hydatid cysts.
- (3) Ovarian cysts, particularly malignant ovarian cysts.
- (4) Solid ovarian tumours.
- (5) A tubal or tubo-ovarian mass.

*The pregnant uterus.*—Usually there is no difficulty in the differential diagnosis between a uterus enlarged by fibroids and one enlarged by pregnancy, but when most of the usual symptoms of either are absent, or the symptoms

are misleading, the difficulties may be great. In such cases it is only by a very careful examination, repeated if necessary at intervals, and by paying careful attention to trivial details, that a correct diagnosis can be reached. Always remember that time will clear up all doubts as to a pregnancy.

Another point to bear in mind is the character of onset and the duration of the symptoms. In pregnancy the symptoms are more or less sudden in onset, the total duration is less than a year, and the rate of growth of the abdominal tumour is uniform and rapid.

Fibroids are much more insidious in onset, the rate of growth is much slower, and the symptoms are usually progressive. The mistake is only likely to occur before the end of the twentieth week of gestation, as after this period the signs of pregnancy become unmistakable. Ballotement is never obtained with a fibroid. If the foetal heart is audible the case is one of pregnancy, but it must be remembered that absence of the foetal heart-sounds in a swelling as large as a twenty-four weeks' gestation does not eliminate a diagnosis of pregnancy; it is only when heard that it is of definite significance, its absence means nothing.

On very rare occasions fibroids are accompanied by amenorrhoea. The condition will invariably be confused with pregnancy until time shows that the tumour is not increasing in size at the normal rate. A woman who is pregnant and wishes to conceal the fact may give a false history of bleeding and omit all concurrent symptoms.

Again, there are cases in which pregnancy does not give rise to amenorrhoea for the first few months or, still more confusing, in which pregnancy threatens to abort at intervals of about three weeks and gives rise to profuse loss and none of the usual symptoms of gestation; this rare hæmorrhagic form of pregnancy is exceedingly difficult to distinguish from fibroids until the foetal heart can be heard.

In this case there appears to be menorrhagia accompanied by enlargement of the uterus — a combination which is typical of fibroids. Careful examination will usually arouse some suspicion, and it is a wise proceeding always to note the condition of the breasts, particularly the nipples and areola. Discoloration of the vagina and blueing of the labia minora are subsidiary signs which may be of great value. The presence or absence of a history of morning sickness is very unreliable either way.



## UTERUS, NEW GROWTHS OF

When any suspicion of pregnancy is present that cannot be cleared up quite satisfactorily on one examination it is well to see the patient again and observe the rate of growth. An enlargement of the uterus accompanied by a *very short history* of bleeding in a woman of child-bearing age should always excite suspicion of pregnancy.

*Abnormal pregnancy* may be still more confusing.

Pregnancy in one horn of a bicornuate uterus may be accompanied by menorrhagia from the other horn and give rise to a misleading train of symptoms. The uterus is enlarged and irregular in outline, and there is no amenorrhœa; in fact it is likely that the loss will be greater than normal owing to the increased blood supply in which the unimpregnated and menstruating half of the uterus shares. In such a case, happily rare, we have to rely on the other signs of pregnancy, particularly the softening of the cervix. The bulk of the tumour may feel cystic and of a consistency which suggests pregnancy rather than a fibroid. The short duration of the symptoms is an important guide, as after the fifth month, if the case be one of pregnancy, it will be more obvious.

Tubal gestation usually gives rise to bleeding at intervals, and the mass formed may be fixed to the uterus. The short duration of the symptoms, together with the severity of other symptoms, will always put one on the right track. The general symptoms of pregnancy are usually well marked in extra-uterine gestation; morning sickness is exaggerated. It is usual for at least one period to be missed before the bleeding begins, and breast changes are usually quite definite. On examination the mass can be felt to be behind the uterus rather than part of it, and is tender to the touch, and much softer than a fibroid.

A still more difficult case is when a hæmatoma formed by an extra-uterine gestation has been present in the pelvis for some months. The tenderness has gone and the clot has become organized and is therefore of firmer consistence.

Cases of this sort may be impossible to distinguish from fibroids unless a history of amenorrhœa at the commencement of the symptoms can be obtained.

*Retroverted gravid uterus.*—The short duration of symptoms and the initial amenorrhœa, together with prominent bladder symptoms, serve to distinguish a retroverted gravid uterus from a fibroid. The initial amenorrhœa is a

most valuable guide. The bleeding is not like the bleeding of a fibroid; the periods have been normal until two or three are missed, then there may be the flooding of a threatened miscarriage, but the bleeding is not of the regular increasing variety associated with fibroids.

On examination, the position of the cervix and the presence of the soft semi-cystic swelling in the pouch of Douglas should offer no difficulties in differential diagnosis.

*Carneous mole.*—A carneous mole may present features almost indistinguishable from a fibroid, especially if one cannot obtain a history of the original pregnancy.

When the history is that the patient thought she was pregnant for four or five months and then the pregnancy seemed to stop, the breasts shrank, and the periods started without any fœtus being passed, and that for the last three or four months the periods have been irregular, with perhaps some sanious discharge in between; and when on examination a uniformly enlarged uterus is found, the presence of a mole should always be suspected. But when no such history is given, either because the patient was unaware she was pregnant, or because she wishes to conceal the fact, we are faced with a patient who complains of excessive loss and on examination we find an enlarged uterus; there are no signs of pregnancy in the cervix, breasts, etc. The tumour does not increase in size if it is left for a month and examined again, so that our sheet anchor for normal pregnancy is lost.

Suspicion may be aroused by the presence of an offensive discharge, especially if this contain bloodclot or altered blood. The short history may put one on the track, and, if suspicion is aroused, an exploration of the cavity of the uterus will give valuable information as to the true nature of the condition.

*Adenoma of the uterus.*—A circumscribed adenomatous growth occurs in the uterus, which may attain considerable size. The symptoms and physical signs are identical with those of submucous fibroids. They may be suspected after removal but can only be diagnosed microscopically.

*Other enlargements of the uterus.*—Malignant disease of the body, as a rule, does not give rise to sufficient enlargement of the uterus to be confused with fibroids. There are, however, two rare forms of carcinoma of the body which cause considerable enlargement. One is the diffuse form in which there is great thickening

## UTERUS, NEW GROWTHS OF

of the whole mucous membrane; whilst in the other there is a patch of carcinoma in the interior of the uterus, and the whole of the peritoneal surface is studded with rounded elevations, which have the appearance of multiple small fibroids, though in reality they are secondary growths. In malignant disease the bleeding is irregular and does not necessarily bear any relation to the periods.

*Pyometra* is accompanied by amenorrhœa unless there is coincident malignant growth, and a purulent discharge is usually present. In *subinvolution* the history of the recent pregnancy and the uniform enlargement of the body together with its slight degree will rarely be confused with fibroids, except perhaps with small ones which are submucous, in which case the same treatment will be suitable as for fibroids.

*Fibrosis uteri*.—Fibrosis of the uterus is met with in two forms. (i) The acute inflammatory form following a gonococcal infection, in which there is a small tender uterus that bleeds excessively at the monthly periods. The uterus is not enlarged, but the condition may be mistaken for a small submucous fibroid if the history of the recent inflammatory disease be overlooked. (ii) The chronic form, occurring chiefly in multiparous women in the late thirties or early forties. The uterus is definitely enlarged and flabby; it is not tender. The bleeding may be so severe as to be almost continuous, and may cause an intense anæmia. This condition can only be distinguished from submucous fibroids by dilating the cervix and curetting the cavity of the uterus, when fibroids if present will become apparent. In many cases the differentiation presents the greatest difficulties.

*Ovarian cysts* may be so tense as to be mistaken for a solid tumour. When this is the case, and more especially when the tumour is adherent to the uterus either from inflammation or from malignancy, the differentiation is wellnigh impossible.

Such a tumour may be accompanied by menorrhagia; the inflammatory reaction of the tissues to which the uterus is adherent causes congestion, and the congested uterus bleeds more easily and more freely. On abdominal examination a firm irregular tumour is felt and the uterus cannot be distinguished separately. Vaginally, there is an irregular mass filling the pelvis—a mass which is either fixed or, if slightly movable, moves the uterus with it.

Ascites is so very rarely found with fibroids that its association with a tumour in the pelvis always strongly indicates a malignant growth, most probably in the ovaries.

*Solid ovarian tumours* are nearly always confused with a subperitoneal fibroid.

Tumours of the ovary in general are associated with scanty periods, so that as a rule the history does not lead one to suspect a fibroid. Ovarian fibromas usually occur at from 18 to 30 years, rather an earlier age than is usual with fibroids. On examination one is struck by the hardness of the tumour. It is freely movable and does not move the uterus. Ovarian tumours tend to lift the uterus out of the pelvis, so that, on vaginal examination, the cervix is high up and can only be reached with difficulty. Uterine fibroids, on the other hand, depress the uterus into the pelvis.

*Tubal disease*.—An inflammatory tubal mass in Douglas's pouch may cause menorrhagia from a congestion of all the pelvic organs, including the uterus. This condition may be of fairly long duration, so that the symptoms resemble those of fibroids to a considerable degree. In salpingitis, however, pain is a symptom especially in the early and acute stage, and the history that the menorrhagia started after an attack of abdominal pain is more likely to suggest tubal disease than fibroids. On abdominal examination it is rare for a tumour to be felt in tubal disease; there is a feeling of resistance, but no definite tumour can be palpated. There is also a certain amount of tenderness varying with the duration and acuteness of the disease.

On vaginal examination a mass is felt which is adherent to the uterus. Here again tenderness is present.

The inflammatory tubal mass is always fixed to the surrounding parts, and therefore more or less immovable in the pelvis. A fibroid may be immovable owing to its filling the pelvis and becoming impacted under the promontory of the sacrum.

Bimanual examination will distinguish between the two, as a fibroid, to become impacted, must have attained a diameter of at least four inches. Fibroids are spherical, unless growth in one particular direction is impeded, so that it is obvious that it will have a longitudinal axis which is as great as or greater than its anterior posterior axis. The mass formed in tubal disease rarely reaches such dimensions, and cannot be felt on abdominal palpation except as a diffuse resistance

## UTERUS, NEW GROWTHS OF

due to the matting of the intestines on its upper surface, whereas a fibroid is felt as a definite tumour. The tubal mass can be felt much more distinctly from the vagina.

A fixed mass which, on bimanual examination, does not appear as a tumour of quite a large size is more likely to be inflammatory than an impacted fibroid.

Tubal disease gives rise to a mass that can be made out to be posterior to the uterus, but this does not distinguish it from a fibroid on the posterior wall.

### TREATMENT OF FIBROIDS

In deciding on the treatment of fibroids many factors must be taken into account and each case judged on its merits. The ideal treatment for any disease is its complete eradication; fibroids can only be got rid of by means of a surgical operation, and there is no doubt that in a large majority of cases an operation is the best and surest treatment.

Fibroids do not always give rise to symptoms; they may be discovered in a patient who is quite unaware of their existence and who has no symptoms referable to the tumour. In such a case we must consider carefully what effect the fibroids are likely to have if they are allowed to remain. On the other hand, a patient may be seen for the first time in an advanced state of anaemia from menorrhagia, or acutely ill with one of the complications described above; in these circumstances there is no doubt that immediate operation is the only treatment.

*Fibroids not causing symptoms.*—The age of the patient is the first consideration. Fibroids delay the onset of the climacteric, menstruation usually not ceasing till the fifty-second year.

There is no doubt that after the menopause fibroids shrink and there is a tendency towards calcification, especially in those that are pedunculated, but on the other hand it is just at this time that certain degenerations and the risk of the development of carcinoma in the myomatous uterus are most to be feared. If therefore fibroids which are not causing symptoms are discovered in a woman in the late forties, the question of subjecting the patient to an operation must be weighed carefully. On the other hand, fibroids in a woman in the early thirties will certainly give rise to symptoms before the onset of the menopause, and it is better to remove them while the patient is in good health than to wait until symptoms, more or less severe, supervene.

*Fibroids and marriage.*—When a woman about to be married is found to have fibroids, even though they are causing no symptoms, the question of the risks that she will run if she should become pregnant is an important consideration.

*Sterility.*—Fibroids are so associated with sterility that their removal by myomectomy offers favourable chances to conception.

*Pregnancy.*—When fibroids are found complicating pregnancy, much care must be given to the question what should be done. Small tumours well out of the pelvis do not as a rule interfere with the gestation and, unless they undergo some complication such as inflammation, degeneration, or torsion, should be left alone. Large tumours and small tumours giving rise to symptoms will have to be removed. The operation of choice in these cases is myomectomy, because it conserves a functional uterus. It is possible in some cases to ligature off or even enucleate the tumour and leave the gestation *in situ*, but as a rule miscarriage will follow. When it is possible to defer the operation until the child is viable, this should be done, so that Cæsarean section can be combined with myomectomy, but when torsion or degeneration (usually of the red variety) forces the surgeon's hand, and the foetus is not viable, it is better to treat the gestation like another fibroid and remove it through the incision for the enucleation of the tumour or tumours. As many as twenty tumours, large and small, can be enucleated in this way and normal pregnancy subsequently occur in the conserved uterus.

If the gestation is likely to go to term, will the fibroids obstruct delivery? If so, Cæsarean section offers a means of saving the child without sacrificing the uterus; at the same time the tumours may be removed by myomectomy.

Fibroids are always liable to degeneration and infection during the puerperium.

*Size.*—The size of the tumours is another important factor. Large tumours, whether giving rise to symptoms or not, are better removed, as it is certain that they will give rise to trouble sooner or later.

*Fibroids causing symptoms.*—The severity of the symptoms is the first consideration. Next comes the age as, to some extent, the nearer the menopause the less urgent the treatment.

When the hæmorrhage has been severe enough to cause anaemia there is no doubt that removal by operation is strongly indicated.

## UTERUS, NEW GROWTHS OF

*Surgical removal cures in toto*; it offers an absolute and immediate cure for all the symptoms. In the hands of an experienced surgeon it is an operation with a general mortality of well under 2 per cent. Hysterectomy offers perhaps less risk than myomectomy on the average, but in our opinion the latter is, in many cases, worthy of consideration.

*X-ray treatment* brings about an artificial climacteric or "change," but in an aggravated form, with the distressing nervous symptoms associated with the abrupt cessation of the ovarian function. It is probable that the sole action of the rays is on the ovaries, it being in fact a bloodless method of spaying the patient. A certain number of cases get relief from the hæmorrhage, and in some cases the tumour shrinks in size as it does after the normal menopause.

The grave objection to X-ray treatment is that it destroys healthy ovaries for the sake of a diseased uterus. It is emphatically not suitable for women who are some years from the climacteric, nor for those who desire children. Its action is slow and uncertain so that it should not be tried when there is definite anæmia, or valuable time will be lost and the patient's chances of successfully enduring an operation will be lessened by the increased anæmia. Those changes in fibroids which are so prone to be associated with the climacteric are anticipated by destroying the ovaries by X-rays.

The difficulty of being absolutely certain that a given tumour is a fibroid is another consideration against the use of X-rays, for we have seen cases of carcinoma of the corpus, ovarian cyst, and inflammatory disease of the appendage treated in this way by mistake.

*Drugs.*—Ergot and hydrastis in their various preparations and forms may be used when it is desired to tide a patient over a short period of time to the onset of the menopause. In some cases they lessen the hæmorrhage by increasing the tone of the uterus. Ergot often gives such severe "contraction" pains that the patients refuse to take it.

Calcium lactate, by increasing the coagulability of the blood, may decrease the loss.

Tonics such as iron, quinine, and strychnine may give some benefit by improving the general health, but these drugs have no real effect on the tumours themselves, and in many cases merely increase the amount of blood lost at the periods.

*Pressure symptoms* can only be relieved by

removal of the tumour; X-rays, drugs, etc., are useless.

*Inflammatory changes*, whether due to torsion of the pedicle or to degeneration, always call for operative interference. In delay there is danger of the inflammation going on to general peritonitis with fatal results.

*Acute torsion* is a catastrophe which demands immediate laparotomy.

### 2. ADENOMYOMATA

Clinically, adenomyomata so closely resemble submucous fibroids that it is impossible to distinguish them; microscopically, they consist of a stroma of connective tissue and, embedded in it, a large amount of smooth muscle-fibres arranged in a network the spaces of which are lined by a columnar epithelium.

The growth may be localized or diffuse. The localized form presents all the appearance of a submucous fibroid except that it does not appear to have a capsule, and it can only be distinguished microscopically. The diffuse form gives rise to a uniform enlargement of the uterus, most of which is due to the growth of the deeper layers of the endometrium. They should be treated by removal.

### 3. CORPOREAL SARCOMA

Sarcoma of the uterus most commonly occurs between the ages of 40 and 60, or in very young girls before puberty.

The commonest form is a sarcoma of the uterine wall. The tumour may consist of round or spindle-shaped cells, and a variety has been described in which the cells showed a cross striation. The clinical features are a soft, rapidly-growing tumour, which bleeds very freely and in general resembles a fibroid. Such a tumour appearing after the menopause must always be regarded with the gravest suspicion.

Sarcomata growing in the body of the uterus have a tendency to become extruded through the cervix, inverting the uterus in the process. So great is this tendency that whenever a sessile or short-stalked tumour has inverted the uterus, a section should always be examined microscopically before deciding whether local treatment is sufficient, or whether radical extirpation of the uterus is indicated.

Another variety described is botryoid sarcoma, which consists of a bunch of grape-like bodies filled with a yellow fluid. They are always more or less polypoid, and hang through the cervix into the vagina.

## UTERUS, NEW GROWTHS OF

### 4. CORPOREAL CARCINOMA

Six types of carcinoma may be met with in the body of the uterus:—

- (1) The localized ulcerative.
- (2) The diffuse ulcerative.
- (3) The localized proliferative.
- (4) The massive proliferative.
- (5) The infiltrating.
- (6) The senile atrophic.

The first five types are columnar-celled carcinomas and do not present any special features microscopically.

The sixth, the senile atrophic type, is not infrequently of the squamous-celled variety. It probably follows on a senile endometritis, in which the columnar-celled ciliated epithelium of the uterus has become transformed into a squamous epithelium. Cell nests are formed.

**Etiology.**—Unlike carcinoma of the cervix, carcinoma of the body does not bear any relation to child-bearing. It is commonest in nulliparous women. The age-incidence is later in life, namely, between the fiftieth and sixtieth years, and it is very rare before the climacteric.

It is frequently preceded by the symptoms of senile endometritis, and is particularly associated with the presence of fibroids in the uterus.

**Pathology.**—The commonest type, the *localized ulcerative*, is characterized by the formation of an ulcer in one part of the body of the uterus, presenting all the typical features of a malignant ulcer. The surface is usually infected with micro-organisms and there is a purulent discharge from its surface.

In the *diffuse ulcerative type* the whole of the interior of the uterus is a raw eroded surface—the cavity is often distended with pus. The walls are thin, and it is obvious that a great part of the muscle has been eaten away by the progress of the growth.

In the *localized proliferative type* there is a projecting mass of growth attached to the uterine wall by its base; it breaks readily on being touched, but is not so grossly septic as the preceding varieties. The uterus is definitely enlarged by its presence.

One of the rarer forms is the *massive proliferative type*. In this it appears that the whole of the endometrium simultaneously takes on malignant characters. The uterus is enlarged. On section the muscle of the wall is thinned out to a mere skin and the whole of the cavity is occupied by the enormously thickened endometrium. The cells are ar-

ranged in regular columns and, microscopically, there is little evidence of malignancy until one examines the edge where the muscle is invaded.

The *infiltrating type* is a rare variety. The whole of the musculature of the uterus is invaded. When exposed at operation the uterus appears as though it were thickly studded with small fibroids, its surface being irregular and covered with small bosses. On section the growth is found to start on the endometrial surface and the bosses on the peritoneal surface to be secondary growths.

The *senile atrophic* form occurs in elderly multiparous subjects. The uterus is small and atrophic, and its peritoneal surface is often crinkled owing to the infiltration of the growth. The growth is very frequently infected with micro-organisms which may lead to the serious complication of pyometra. Pus may leak along the Fallopian tubes into the peritoneal cavity, causing pelvic peritonitis, and may carry with it particles of growth which may become implanted on the peritoneum.

**Symptomatology.**—The symptoms are typical in the majority of cases. They usually come on at, or several years after, the menopause. A patient who has had no periods for a few years complains that she had a slight show for a few days, then no bleeding for a time, and then a flooding. All may subside for a time and then another flooding occurs.

On examination the cervix is quite healthy. Although there is frequently some enlargement, the uterus is not uncommonly small and atrophic.

The degree of mobility depends on the stage to which the disease has progressed and whether there has been any perimetritis.

**Diagnosis.**—In all cases in which there are symptoms of bleeding after the menopause the uterus should be dilated so that its interior may be explored. This is not a very difficult proceeding. The instruments required are volsella forceps (two pairs), a uterine sound, a set of graduated dilators, a curette, and a speculum, such as a Sims' duck-bill or an Auvard's self-retaining vaginal speculum, to keep the vagina open.

The patient is anæsthetized after a preliminary antiseptic douche and placed in the lithotomy position in a good light. The speculum is introduced and a good view of the cervix obtained. If there has been any septic discharge the vagina can now be swabbed out with an antiseptic solution and the vagina

## UTERUS, NEW GROWTHS OF

and cervix painted with iodine. The cervix is next seized with volsella forceps and pulled down. It is better to apply a second pair of these forceps to steady the cervix. The sound is gently inserted and passed into the cavity to ascertain its size and direction; in this manoeuvre the utmost caution must be used, as it is extremely easy to perforate the uterus and so infect the peritoneal cavity, with disastrous results.

The dilators are inserted in graduated sizes until the cervix is sufficiently dilated to admit the curette. The curette is then passed and the cavity of the uterus gently curetted; a softened area which bleeds freely is diagnostic of carcinoma.

Any piece curetted out should be sectioned and examined microscopically.

**Treatment.**—If cancer be present, total hysterectomy with wide removal of the ovario-pelvic ligaments is the only treatment.

Cancer of the body of the uterus spreads with relative slowness, and is late in giving rise to metastases. The prognosis, therefore, is good, especially in those cases in which the growth is associated with large fibroids.

### 5. CERVICAL CARCINOMA

Carcinoma of the cervix is very uncommon in women who have not borne children, and most extremely rare in women who have never had sexual intercourse. The common age-incidence is between 40 and 50 years, but cases have been reported in patients under 25.

There are two histological varieties:

Columnar-celled carcinoma.

Squamous or spheroidal-celled carcinoma.

Of these the second accounts for at least 96 per cent. of the cases.

**Clinical types.**—Clinically, carcinoma of the cervix may be described according to the type of growth which takes place, and according to the situation in which it arises. The growth may be ulcerating, fungating, infiltrating, or massive in type; there is also an atrophic senile type.

Carcinoma may begin on the vaginal surface of the cervix, or just at the external os, or in the cervical canal itself.

The *ulcerating type* is an ulcer almost from the start. It commences as a small raised papule, which rapidly breaks down in the centre, leaving a shallow ulcer with raised hard edges and an excavated friable base.

The *fungating type* either protrudes through the cervix like a mushroom or, if growing on

the vaginal portions, fills the vault of the vagina with a cauliflower-like mass.

The *infiltrating* or *massive type* begins in the cervical canal and infiltrates the whole cervix without breaking down, so that, on section, the cervix is a hard solid mass. The cervical canal may be occluded and the cavity of the uterus distended with pus and blood—pyometra. Pus may leak along the Fallopian tubes, setting up pelvic peritonitis.

The *senile atrophic type* gives rise to few signs. The vaginal portion of the cervix is shrunk and atrophied, and the vault of the vagina puckered, and through the little depression, which represents the external os, blood may be seen to come. The uterus is small.

**Symptomatology and diagnosis.**—It must be insisted that flooding, intermittent hæmorrhage, and sanious or foul discharge are not symptoms of the normal "change." All excessive hæmorrhage, at or about this time, be it a sudden flooding or a long-drawn-out intermittent losing, should be looked upon with very grave suspicion; it is imperative in all cases that a vaginal examination be made. If nothing abnormal is found and the bleeding continues, a second examination must be made, during which it is advisable to administer an anæsthetic, to dilate the cervix, and to explore the interior of the uterus and cervical canal.

In nearly all cases of carcinoma of the cervix the very first symptom is bleeding—usually a fairly copious loss at about the time a menstrual period is due. The bleeding may cease after a day or two and be followed by amenorrhœa for two or three weeks, after which there is either another flooding or a slight daily loss extending over a period of days or months with one or two floodings interspersed between. The loss in the intervals between the floodings nearly always becomes offensive and may contain shreds of broken-down tissue.

Bleeding is increased by examination and by coitus; indeed, bleeding after coitus is not uncommonly one of the first symptoms.

The signs differ according to the variety. In the *infiltrating type* the cervix is felt to be enlarged, thickened, and hard. The vaginal surface of the cervix may feel normal, so that it is only when a sound is passed into the cervical canal that the carcinomatous nature of the growth is apparent. The same is true of all intracervical growths in the early stages; hence the importance of examining the cervical canal in every case with a suspicious history.

## UTERUS, NEW GROWTHS OF

The *ulcerating type* appearing at the external os, or on the vaginal surface of the cervix, is easily recognized. On vaginal examination an ulcer is felt unless the growth is an early intra-cervical one. It has a typical feel, with the hard indurated edges and irregular friable base characteristic of all malignant ulcers. The surface of the ulcer has been described as feeling like wet indiarubber.

On withdrawing the examining finger it is extremely important to note the presence of a dirty bloodstained discharge; this is constantly present and is diagnostic.

The cervix is much enlarged and hard, and its mobility is much impaired.

Pain and wasting are both late symptoms. The cervix is very poorly supplied with nerves of sensation, especially that of pain, and volsella forceps can be applied without the patient being conscious of its presence; therefore, when a carcinoma of the cervix gives rise to pain, it is an indication that the disease has spread beyond the cervix.

Valuable information can be obtained from a visual examination of the cervix, and it is a wise proceeding to make a practice of inserting a speculum and looking at the cervix in a suspicious case; the appearance of a malignant ulcer seldom leaves any doubt as to its nature. A growth which is still intracervical can frequently be detected by examining the cervical canal with a probe. When a probe is passed gently into the cervical canal of a healthy uterus there may be a few drops of blood, but the bleeding cannot be confused with the copious ooze of dark blood which comes from a cancer.

The *fungating type* is not likely to escape detection. The soft friable mass projecting into the vagina, which breaks away on being touched, and bleeds readily, is so characteristic that it needs no further description.

The *senile atrophic form* is very difficult to diagnose, since the symptoms are not pronounced, and the small cervix and uterus are deceptive.

In all cases of doubt a portion should be cut from the cervix under an anæsthetic and examined microscopically.

**Method of spread.**—The growth commences as a downgrowth of the epithelium of the surface or lining a gland. Its rate is slow, metastases are formed late, and in the early stage it is limited to the cervix itself. Thence it spreads downwards into the vagina, forwards to the base of the bladder, backwards to the

peritoneum on the posterior surface of the cervix and so to the rectum, and laterally into the broad ligaments. Metastases first occur in the glands in the obturator fossa, later in those along the external iliac vein, and later still in the lower aortic glands.

Carcinoma of the cervix is nearly always infected with septic organisms, and the consequent inflammation may spread along the lymphatic tracts in advance of the growth, so that the broad ligaments and glands appear to be infiltrated with growth when in reality they are only indurated by inflammation.

**Prognosis.**—Carcinoma of the cervix, when diagnosed early, is one of the most satisfactory of all cancers as regards the results of radical operation. Recurrence at a distance is rare even in advanced cases, and, provided that the growth has not spread beyond the uterus and broad ligaments, the disease can be extirpated completely. The operation is a severe one, and increases in severity with the advance of the growth. The results, in the hands of experts, have very greatly improved during the last few years.

**Criteria of operability.**—The question whether a case is operable or not is one of extreme importance. The points to be ascertained before arriving at a conclusion are:

(1) *Duration of symptoms.*—As a rule, the rate of growth is slow, and in average cases, if symptoms have been present for no more than three months, the case is certainly operable, for six months it is probably operable, for nine months it is possibly operable, though probably not, for a year or over it is most likely inoperable.

(2) *Length of anterior vaginal wall.*—It is easy to mistake the hard edge of an ulcer extending all round the vagina for the cervix, and one may be misled into thinking that the growth is confined to the cervix when in reality it has invaded the greater part of the vagina. In advanced cases there is usually much apparent shortening of the anterior vaginal wall as measured from the margin of the ulcer or growth to the vaginal orifice. When the growth has extended on to the anterior vaginal wall it is highly probable that the base of the bladder is involved.

(3) *Bladder symptoms.*—Either painful or frequent micturition is an unfavourable sign, indicating infiltration of the bladder-wall.

(4) *Urine.*—Examination of the urine will give valuable evidence of the extent of the growth anteriorly. The presence of pus and

## UTERUS, NEW GROWTHS OF

blood, especially of the latter, is strong evidence that the bladder is hopelessly invaded.

(5) *Cystoscopic examination of the bladder* will show whether the growth has reached its mucous membrane.

(6) *Pain*.—Severe pain is indicative of inoperability, for so long as the disease is confined to the cervix pain is seldom complained of. Pain on micturition or defaecation indicates involvement of the bladder or the rectum.

(7) *Fixity*.—Apparent fixation of the cervix does not always indicate inoperability. As pointed out above, the inflammatory reaction may spread much more rapidly than the carcinoma. If the fixation is due to inflammation the growth can probably be got away, and there is a fair chance of avoiding recurrence.

The amount of fixation can be tested by pressing the finger firmly between the growth and the pelvic wall; if there is any "give" here, the growth is probably operable.

Fixation to the rectum, as evidenced by the induration of the posterior fornix, is an unfavourable sign. Rectal examination will often reveal the extent to which the rectum is involved. In doubtful cases the abdomen should be opened, for this is the only way of deciding whether the growth is removable or not.

**Treatment.**—Radical extirpation by surgical means is desirable if it be possible. The operation, which was placed on a sound basis by Wertheim, offers a 50-per-cent. chance of permanent cure to those who survive it. The operative mortality varies according to the stage of the growth. In early or moderately advanced cases it is somewhere about 7 per cent., or less. With advanced cases, although the operative mortality is necessarily high (15 to 20 per cent.), hope need not be abandoned, for many cures are on record even when the removed obturator and iliac glands were found to be full of malignant growth. The operation, which is exceedingly difficult and requires special experience and skill, is one of the great triumphs of surgery.

**Palliative treatment of inoperable growths.**—It is important to bear in mind that sepsis plays a very important part in the pain and hæmorrhage which are such distressing symptoms of the late stages of this disease. The offensiveness of the discharge is also attributable to sepsis.

Treatment by *radium* has given very unequal results. Radium rays exert an effect

mainly through the formation of fibrous tissue of such density that it appears to strangle the soft cancer cells enclosed in its meshes, so that what was a soft friable ulcer, bleeding on the slightest provocation, may become a hard indiarubber-like mass which offers marked resistance to the scalpel, and appears white and bloodless on section. Micro-organisms appear to be killed by the action of the rays, so that the discharge is lessened. The treatment is frequently followed by a good deal of pain due to the fibrous tissue—which is formed for a distance of about 3 cm. from the point of application—contracting and involving nerves in the process. The pain may pass off to some extent, and is then more than counterbalanced by the temporary freedom from discharge and bleeding. A few cases are on record in which radium appears to have effected a cure, but in general the best that can be said of it is that it greatly improves some cases for a shorter or longer period, while on the other hand some cases are undoubtedly the worse for it.

Other forms of treatment include those which are local, such as antiseptic douches, scraping, and cauterizing.

When there is a large fungating growth, temporary benefit follows scraping and cauterization. An anæsthetic is necessary, and the patient is placed in the lithotomy position. The vagina is douched and swabbed out. A large portion of the growth can often be scooped out with the fingers, but the operation should include scraping with a curette and should be completed by cauterizing the raw surface, preferably with the actual cautery; swabbing with pure carbolic acid is permissible.

Douching twice daily with hypochlorite solution or with tinct. iodi mitis or lysol, 1 dr. to the pint of either, is useful for lessening the discharge and odour.

For hæmorrhage, peroxide of hydrogen is more useful, and it may be necessary to plug the vagina.

### 6. SARCOMA OF THE CERVIX

Sarcoma of the cervix is rare and is indistinguishable from carcinoma except with the aid of the microscope. The symptoms, signs, and treatment are identical with those of carcinoma. The prognosis after operation is not quite so good, as the growth tends to disseminate earlier.

VICTOR BONNEY.

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## UTERUS, POLYPI OF

**UTERUS, POLYPI OF.**—Polyps of the uterus may be—

1. Fibroid.
2. Mucous.
3. Placental.
4. Malignant.

### 1. FIBROID POLYPI

When a small submucous fibroid is present in a uterus, it is regarded as a foreign body, and by a series of peristaltic waves of contraction the uterus attempts to expel it. These efforts are frequently successful and the tumour is dragged away from the uterine wall but remains attached by a stalk containing the blood-vessels. The growth now constitutes what is known as a fibroid polyp.

The uterus continues its expulsive efforts, and eventually pushes the tumour through the cervix into the vagina. Thus is produced the condition usually seen when a patient is suffering from a fibroid polyp.

In the vagina, just outside the cervix, is an oval-shaped body of firm consistence attached to a stalk which passes up into the uterus. It may vary in size from a hazel nut to a foetal head at term.

Although these tumours arise in the body of the uterus, their point of attachment may migrate towards the cervix, so that it not infrequently happens that the stalk is attached to the edge of the cervix itself.

When they are of comparatively large size, the broad base offers such resistance, to being stretched that it drags the fundus of the uterus down with it. The fundus is then gripped by the sides of the uterus and the whole uterus may be turned inside out and be found in this condition hanging in the vagina with the fundus, surmounted by a tumour, presenting at the vulva.

At other times, a large tumour may be extruded through the cervix, and by its weight may drag the uterus down, causing prolapse. This form of prolapse does not lead to prolapse of the vaginal walls and therefore is unaccompanied by cystocele or rectocele.

**Symptomatology and diagnosis.**—The symptoms of fibroid polyps are first of all those of the submucous fibroid from which they arise. Being usually of small size, they do not cause symptoms by pressure.

For a year or two there is menorrhagia, with some pain at the periods and leucorrhœa between them. While the polyp is being extruded there is usually an exacerbation both

of the bleeding and of the pain, but once it has escaped through the cervix the pain ceases, the leucorrhœa is usually increased, and the hæmorrhage becomes irregular, the polyp bleeding on the slightest provocation. Polyps usually bleed on examination and frequently after coitus.

Per vaginam, the finger comes on a rounded lump, which is freely movable, though small polyps are sometimes difficult to feel, as they slip away from the finger. The finger is passed on around the tumour, and the cervix can be felt with the thin stalk of the tumour coming through it.

If it is no bigger than a walnut the diagnosis can be absolutely clinched by inserting a speculum and seeing the tumour and the cervix together.

When the tumour is larger this cannot be done, and care must be taken to differentiate between a polyp and a chronically inverted uterus, or a fibroid inverting the uterus. This is an important point, as removal of a tumour through its pedicle, when that pedicle happens to be the body of an inverted uterus, would be disastrous.

A polyp has a narrow stalk; an inverted uterus, whether simple or complicated by a submucous fibroid, has a broad stalk, i.e. the body of the uterus. The stalk of a polyp is gripped by the cervix, which closely invests it; an inverted uterus has the cervix spread out like a band round the upper part of the tumour.

A sound should be passed into the cavity of the uterus following up the stalk of the tumour. In the case of a polypus it passes to the normal length or further (the body of the uterus may be enlarged from congestion); in a case of partial inversion it passes only a short distance. In inversion it is sometimes possible to demonstrate the orifices of the Fallopian tubes.

A rounded tumour in the vagina may be a fibroid sessile on the vaginal portion of the cervix. In this case the cervix is found to be continuous with the tumour and to one side of it. There is no stalk and the tumour is not freely movable.

**Pathology.**—The core is formed by a fibromyoma similar in every respect to those found in the uterus; there are spindle-shaped cells with small nuclei arranged in whorls and with a scanty amount of connective tissue and blood-vessels. The tumour is invested in a covering of uterine endometrium, except where

## UTERUS, POLYPI OF

it has been subjected to friction; here the cilia are worn off and the columnar become changed into squamous cells. Beneath the mucous membrane is a layer of connective tissue and muscularis mucosæ; dipping into this a few glands may be seen, of the simple tubular type, resembling those of the uterus.

The pedicle consists of a core of blood-vessels supported by a small amount of connective tissue and covered with mucous membrane of a similar type. The whole of the tumour is ordematous, partly from congestion due to the constriction round its pedicle and partly from inflammation by organisms of the vagina.

**Complications.**—The complications are sepsis, sloughing, and malignant disease.

There is no definite evidence that a simple polyp becomes malignant, and it must be borne in mind that some of these tumours are really sarcomatous from the outset.

When one is extruded, even only partially, through the os, it becomes infected by the various organisms present in the vagina. In most cases this simply causes it to become generally ordematous, but sometimes the infection is so acute as to cause sloughing, accompanied by symptoms of general toxæmia and a foetid discharge from the vagina; the discharge may contain shreds of disintegrated tissue. The sepsis may be so severe as to cause death unless dealt with promptly.

**Treatment.**—The treatment of uterine polypi, except when they are large in size and fill the vagina, is simple.

The bowels should be emptied a few hours before operation and a vaginal douche given of some antiseptic solution, such as tincture of iodine or lysol, 1 dr. of either to the pint. The patient must be anæsthetized and placed in the lithotomy position in a good light.

A vaginal speculum is necessary, preferably one of the Auvard's self-retaining type. A Sims duckbill speculum can be used satisfactorily, but requires an assistant to keep it in place so that the operator may have both hands free.

Small tumours with a thin pedicle may be grasped with notched forceps and twisted round and round, without any traction being made, until the pedicle breaks through. This procedure is rarely followed by bleeding. Slightly larger tumours with a thicker pedicle are not so simply treated; it is advisable to ligature the pedicle and cut the tumour away.

To carry out this operation, follow the stalk

as far up the cervical canal as practicable. The tumour should be pulled down, to bring more of the pedicle within reach. The pedicle is then grasped with Spencer Wells forceps, and cut through distally to the forceps. A ligature is applied and tightly tied proximally to the forceps and the forceps removed.

Large tumours may so fill the vagina that it is impossible to get at the pedicle at all. They must be removed piecemeal, a very difficult operation which should only be attempted by the expert.

Sessile tumours dragging down the fundus of the uterus are still more difficult and dangerous.

### 2. MUCOUS POLYPI

Occasionally small soft polypoid growths are found hanging from the cervix or are scraped out of the uterus during the operation of dilating and curetting. These are known as mucous polypi. They are probably formed either by little sessile overgrowths of mucous membrane, which become polypoid in the same way as fibroids, or they are the end results of incomplete separation of strips of mucous membrane during menstruation or parturition.

These polypi are rarely found with long stalks. Those which appear at the external os usually arise just inside the cervical canal. They are generally about the size of a pea or horsebean, and bright red in appearance.

**Symptomatology.**—The symptoms of a mucous polyp are excessive bleeding and leucorrhœa. When projecting through the os, it forms a soft tumour about the size of a pea or a marble, which is difficult to palpate, as it slips away from the examining finger. Viewed through a speculum it is quite unmistakable, and appears as a bright red spot just protruding through the os.

Mucous polypi and placental polypi which remain in the cavity of the uterus give rise to excessive bleeding at the monthly period and leucorrhœa in the intervals, and can only be diagnosed with certainty when the cervix has been dilated, and they are either felt with the finger or curetted out.

**Pathology.**—The growths consist of mucous membrane covering a core of connective tissue. For treatment, *see* below.

### 3. PLACENTAL POLYPI

During the third stage of labour it sometimes happens that a small portion of placenta remains attached to the wall of the uterus. This piece of the placenta may not become

## UVEAL TRACT, AFFECTIONS OF

infected and may continue to receive nourishment from the blood-vessels of the uterus; it then forms what is known as a placental polyp.

**Treatment.**—The treatment of placental as of mucous polypi consists in dilating the cervix and curetting the polypi out with a sharp curette. The patient must be prepared and anæsthetized and placed in the lithotomy position. A speculum is introduced into the vagina and the cervix seized with volsella forceps. Graduated dilators are passed until the cervical canal will admit the finger. The interior of the uterus can then be explored and any polypi are curetted away. The cavity should be washed out with an antiseptic solution. It is not usually necessary to pack the uterus, as the bleeding soon stops.

### 4. MALIGNANT POLYPI

A sarcoma growing in the uterus has a tendency to become polypoid; such a tumour may be mistaken for a fibroid polypus, but recurs after removal. Should recurrence take place after a supposed fibroid polypus has been removed, the tumour should always be examined microscopically for sarcoma.

VICTOR BONNEY.

LOUIS CARNAC RIVETT.

**UTERUS, PROLAPSE OF** (*see* PELVIC ORGANS, FEMALE, DISPLACEMENTS OF).

**UTERUS, RETROVERTED GRAVID** (*see* RETROVERTED GRAVID UTERUS).

**UTERUS, RUPTURE OF** (*see* LABOUR, MATERNAL INJURIES FOLLOWING).

**UTERUS, SUBINVOLUTION OF** (*see* SUBINVOLUTION OF THE UTERUS).

### UVEAL TRACT, AFFECTIONS OF.—

In this article are included—

1. IRITIS.
2. IRIDOCYCLITIS or CYCLITIS.
3. SYMPATHETIC OPHTHALMIA.
4. CHOROIDITIS.

#### 1. IRITIS

It must be remembered that inflammation of the iris is always accompanied by some degree of inflammation of the ciliary body (cyclitis), and that sometimes the choroid also is affected.

The iris is practically a diaphragm of blood-vessels, which run a radial course, unstriped muscle-fibres, and a loose stroma. When it becomes inflamed the blood-vessels dilate and

exudates are poured out into the lymph spaces and into the anterior chamber. Dilatation of the vessels causes constriction of the pupil, which is increased by the irritation of the sphincter pupillæ. The exudation into the stroma obscures the iris pattern, so that it becomes blurred and "muddy." It impedes the pupillary movements, which become sluggish. The muddiness is associated with change in colour, most marked in blue irides, which become greenish; brown irides tend to become greyish or yellowish-brown. Since iritis is usually unilateral during the acute phase, the difference in colour of the two eyes is an important diagnostic sign.

The hyperæmia also affects the anterior ciliary vessels, so that there is a zone of deep-seated congestion around the cornea. The conjunctival vessels are also somewhat engorged, and care must be taken to distinguish the condition from conjunctivitis. There is watering of the eye, but no muco-purulent discharge.

From irritation of the sensory nerve-fibres there is severe neuralgic pain. Pain is also referred to other branches of the ophthalmic division of the fifth nerve, notably in the brow and over the malar region. It is specially severe at night.

The exudates in the anterior chamber cause the aqueous to become cloudy, which may be mistaken for haziness of the cornea. In cases due to virulent pyogenic organisms, whether of ectogenous or endogenous origin, the aqueous contains polymorphonuclear leucocytes, which sink to the bottom of the anterior chamber and form a hypopyon. This is rare in primary iritis, as is also blood in the anterior chamber (*hyphæma*).

The exudates form a thin film over the anterior surface of the iris and spread over the pupillary margin, sometimes covering the pupillary area. They cause adhesion of portions of the margin of the pupil to the lens capsule (*posterior synechia*). In the early stages the adhesions are not very firm, and if the pupil is dilated with atropine they break down, often, however, leaving specks of pigment upon the lens capsule. These fragments of iris pigment persist for a very long time, and are valuable evidence of a previous attack of iritis. The exudates tend rapidly to organize and form permanent adhesions. In this state atropine causes the pupil to dilate irregularly, only the intervening free portions becoming retracted, so that the pupil assumes a festooned appearance.

## UVEAL TRACT, AFFECTIONS OF

In severe cases, especially if a mydriatic has not been used early, and after recurrent attacks, the whole of the pupillary margin may become adherent to the lens capsule (*ring synechia* or *seclusio pupillæ*). Organization of exudates in the pupillary area leads to the formation of a film of fibrous tissue (*blocked pupil* or *occlusio pupillæ*). If the ciliary body is much involved in these severe plastic cases the whole of the posterior surface of the iris is tied down to the lens capsule (*total posterior synechia*). The peripheral part of the iris is then drawn back, and this part of the anterior chamber becomes abnormally deep.

Mild cases of acute iritis may subside in three or four weeks, but cessation of treatment will often be followed by recrudescence. One of the most characteristic features of iritis is the tendency to relapse, each attack, though usually less severe than its precursors, leading to more adhesions and often more impairment of vision. In the more chronic cases the ciliary body is generally more seriously involved (*iridocyclitis*, p. 480).

If no mydriatic is used synechiæ form and the case becomes grave, for vision will probably be impaired and fresh attacks will increase the adhesions. Eventually in such a case a ring synechia is likely to be formed. This, unless treated by operative measures, will inevitably lead to secondary glaucoma, for the fluid secreted by the ciliary body is dammed up behind the iris, which becomes bowed forwards like a sail (*iris bombé*).

Repeated attacks of iritis lead to atrophy of the iris, which becomes grey like blotting-paper, and may actually have holes in it.

**Diagnosis.**—Acute iritis is most frequently mistaken for *acute glaucoma*, a very serious error, since the treatment of the two conditions is diametrically opposed. The chief distinguishing features are: (1) In iritis the pupil is small and irregular; in acute glaucoma it is large and oval. (2) In iritis the intra-ocular pressure is normal or very slightly raised; in acute glaucoma it is raised and is often very high. (3) In acute glaucoma the pain is sudden in onset and is often accompanied by vomiting; in iritis it is neuralgic in character and worse at night. (4) Vision is usually more impaired in glaucoma than in iritis.

**Varieties.**—The chief varieties of iritis are syphilitic, gonorrhœal, "rheumatic," diabetic, tuberculous, sympathetic (see *Sympathetic Ophthalmia* p. 480), and purulent (see *PAN-OPTHALMITIS*).

**Syphilitic iritis** is the commonest form of plastic iritis. It usually occurs within a year of infection, and in at least 3–4 per cent. of syphilitics. It is generally unilateral, but the second eye becomes affected in about a quarter of the cases. It differs from "rheumatic" iritis in that it shows little tendency to recur. In cases of doubt the Wassermann test should be applied. There are very severe forms of gummatous iritis in which yellowish-red nodules form near the pupillary and ciliary borders of the iris; they are usually multiple. There is much exudation in these cases, and, exceptionally, broad synechiæ are formed. Syphilitic iritis is a sign of severe infection.

**Gonorrhœal iritis** is common, and is associated with gonorrhœal "rheumatism"; it tends to recur. Both eyes are affected, but seldom at the same time. Not infrequently the exudate in the anterior chamber has a characteristic gelatinous appearance.

**"Rheumatic" iritis** is usually associated with rheumatic pains in the joints and muscles. Iritis is rare in true acute rheumatism. Probably most cases are due to septic absorption from some focus in the body. Other patients are gouty or have rheumatoid arthritis. "Rheumatic" iritis is usually of moderate severity, attacks both eyes, and shows a marked tendency to recur. Gouty iritis occurs in elderly patients, often starting suddenly in the night, and sometimes ushering in an acute attack of gouty arthritis.

**Diabetic iritis** is rare. New and enlarged vessels are sometimes formed on the iris, and there is occasionally a hypopyon.

**Tuberculous iritis** may be of the miliary or conglomerate type. The nodules in the iris are at the pupillary or ciliary borders. The condition resembles gummatous iritis, but is usually quieter.

The **treatment** of iritis is local and constitutional. The pupil should be dilated with atropine as rapidly as possible; guttæ or ung. atropinæ (1 per cent.) should be used at least every four hours until the pupil is well dilated. Hot fomentations should be employed to relieve pain and to assist in dilating the pupil. In recalcitrant cases two or three leeches to the temple have an extraordinarily beneficial effect on the pain and inflammation. Aspirin or even morphia may be given to relieve the intense pain.

General treatment should be begun with a strong saline purge. Syphilitic cases respond well to mercury and iodides, and also to

## UVEAL TRACT, AFFECTIONS OF

salvarsan and its congeners. In other types of iritis, salicylates often do good, probably by acting as intestinal disinfectants. Atropine must in all cases be continued for a week or fortnight after the eye appears to be quiet, otherwise a relapse is very likely to occur.

If a ring synechia forms, an iridectomy must be done, but should not be undertaken until the acute stage has subsided. An iridectomy is indicated in order to prevent a ring synechia forming when there are numerous posterior adhesions, the result of recurrent attacks of iritis.

### 2. IRIDOCYCLITIS OR CYCLITIS

These terms denote inflammation of the iris and ciliary body in which the main stress falls upon the latter. The clinical features differ from those of iritis (q.v.), in which the iris is chiefly involved.

**Acute iridocyclitis.**—In the acute form exudates are poured out freely from the ciliary body. They surround the lens and extend throughout the vitreous. By organizing they cause total posterior synechia (*see* Iritis, p. 479), and cyclitic membranes in the vitreous. Owing to interference with nutrition the lens becomes cataractous. The bands in the vitreous contract and pull up the retina, causing detachment of the retina and so-called shrinking of the vitreous. Eventually the ciliary processes are destroyed and the secretion of lymph is seriously diminished. The tension of the eye falls, the structures succumb to malnutrition, and the eye shrinks (phthisis bulbi). In these shrunken globes the choroid becomes converted into bone.

**Chronic iridocyclitis** (simple cyclitis or "serous iritis") is a very insidious disease. Vision diminishes without obvious cause. Minute investigation, however, reveals tenderness of the eyeball, especially over the ciliary region, readily induced ciliary congestion and irritation on exposure to light, etc., and dust-like opacities in the vitreous. Most characteristic are tiny grey spots on the back of the cornea (keratitis punctata or "k.p."). The spots consist of leucocytes deposited from the aqueous upon the back of the cornea, where they stick. In the typical condition they are scattered over a triangular area of the lower part of the cornea, the smaller spots above, the larger below. Often only a few spots are found irregularly distributed. They may be pigmented, in which case the pigment persists long after the inflammation has subsided. The smaller spots require strong illumination and

magnification with a corneal loupe for their discovery.

Posterior synechiæ are not a conspicuous sign of iridocyclitis, but are liable to form insidiously. The anterior chamber is deeper than normal, probably owing to the difficulty of filtration out of the eye of the more highly albuminous aqueous formed under these conditions. For the same reason the intra-ocular pressure is somewhat raised, and may be so high as to demand operative interference in order to save the retina and optic nerve from its effects.

Iridocyclitis is very liable to recurrences, each of which is likely to cause further permanent impairment of vision. In these cases the iris often shows signs of atrophy—obscuration of the iris pattern and change in colour.

Chronic iridocyclitis is commoner in women than in men, and is probably always due to some toxæmia or bacterial metastasis. Careful search for some septic focus should be made—in the mouth (pyorrhœa alveolaris), nose, and accessory nasal sinuses, alimentary canal, generative organs, etc. Syphilis or tubercle is more rarely the cause.

The **treatment** is essentially the same as that of iritis (q.v.). Locally, atropine and hot bathings are indicated in the acute stage; rest and dark glasses in the later stages. Any possible source of toxins must be treated. In a large proportion of cases the search fails. In them small doses of calomel ( $\frac{1}{4}$  gr. three times a day) are often found particularly beneficial; or salicylates may be given. Iodides help to bring about absorption of exudates and vitreous opacities. In the worst cases the patient should be kept in bed and submitted to mercurial inunctions. Vapour baths and pilocarpine injections have proved useful in some cases.

If the tension is seriously raised it must be relieved by paracentesis, which has, however, only a transitory effect. Repetition every two or three days will often induce a healthier condition of the ciliary body and lead to more permanent relief of tension. The more active lines of treatment should be followed by tonics, good diet, and plenty of fresh air.

### 3. SYMPATHETIC OPHTHALMIA

Sometimes the sound eye (sympathizing eye) is attacked by serious inflammation when the other (exciting eye) has been injured. The injury to the exciting eye is usually a perforating wound, and it is probable that sympathetic

## UVEAL TRACT, AFFECTIONS OF

ophthalmia does not occur unless the walls of the globe have been pierced. Wounds in the ciliary region, with incarceration of the iris or ciliary body, are undoubtedly the most dangerous. Whether the retention within the eye of a foreign body causes an additional danger of this disease is much more doubtful. If the foreign body is aseptic and is not lodged in the ciliary body it is probably innocuous from this point of view. If suppuration occurs, sympathetic ophthalmia rarely, if ever, follows. Hence it is not caused by perforating corneal ulcers.

Sympathetic ophthalmia may occur at any age, but children are specially susceptible. It almost always supervenes in from three to eight weeks of the receipt of the injury.

There is invariably severe iridocyclitis in the exciting eye. Marked tenderness, lachrymation, ciliary injection, and, above all, precipitates ("k.p.") (see Iridocyclitis, p. 480) on the back of the cornea are grave signs of danger to the other eye, especially if the iris or the ciliary body is incarcerated in the wound and the eye shows no signs of quieting down. Sometimes the inflammation causes the injured eye to shrink, and it may long remain quiescent, yet sympathetic ophthalmia may occur. In these cases it is usually preceded by a return of acute symptoms in the exciting eye.

The iridocyclitis of the sympathizing eye is usually of the plastic type. If careful watch has been kept upon this eye the first sign is usually the appearance of spots of "k.p." on the back of the cornea, often accompanied by a ciliary blush, and some irritability, shown by photophobia and readily induced lachrymation. In neglected cases the patient first notices defective vision. Near objects become blurred, but after a rest vision improves. This is due to weakness of accommodation.

Milder cases may without apparent reason assume a more dangerous form. Those which show little exudation from the iris and ciliary body have a more favourable prognosis. Here there are the signs of a simple iridocyclitis, with deep anterior chamber, "k.p.," little tendency to the formation of synechiæ, and relatively little ciliary injection. In these circumstances the tension may be slightly raised, and raised tension is more to be desired in these cases than subnormal. In the more severe plastic type the pupil becomes quickly blocked with exudates, posterior synechiæ are rapidly formed, and exudates are poured out by the

ciliary body, leading to lowered intraocular tension and secondary cataract. In cases of intermediate severity a ring synechia and secondary glaucoma may occur.

Sympathetic ophthalmia is probably due to general infection through the blood-stream by a specific organism introduced into the exciting eye.

The **treatment** varies according to the case, and often demands the exercise of great judgment. Sympathetic ophthalmia never occurs if the injured eye is excised before the second eye is attacked. Hence, if the injured eye is so damaged that no useful vision can be expected with it, risk should be avoided by removing it. If, however, good vision may reasonably be expected, expectant treatment should be adopted for a time. If the eye quiets down quickly, no harm is likely to accrue. If the iris, ciliary body, or lens capsule is incarcerated in the wound, every effort should be made to free it. The prognosis is better if the lens is not wounded. The iris and ciliary body should be kept at rest by atropine. If in spite of all efforts the eye remains irritable, and especially if "k.p." appears, the eye should be excised. The slightest sign of ciliary injection or "k.p." in the other eye indicates the immediate necessity for excision of the injured eye.

Still more difficult is the decision when sympathetic ophthalmia has already started and the injured eye has useful vision. If the inflammation in the sympathizing eye is severe it may be lost in spite of all efforts. Moreover, excision of the exciting eye has little beneficial effect on the inflammation in the other eye if it has already taken firm hold.

Otherwise, the treatment is that of iridocyclitis in general (p. 480). In the later stages pilocarpine injections and iodides by the mouth assist the absorption of exudates. The results of treatment are usually disappointing. Improvement of vision may sometimes be obtained by an optical iridectomy, but this must not be undertaken until the eye has been quiet for several weeks or months. In severe cases, if perception of light persists, more desperate operations, such as extraction of the lens, etc., may justifiably be undertaken if the other eye is blind or has been removed.

### 4. CHOROIDITIS

This term is used to include all inflammatory and degenerative changes in the choroid. Owing to the dependence of the outer layers

## UVEAL TRACT, AFFECTIONS OF

of the retina for their nutrition on the integrity of the choroid, the retina always suffers in these cases, and the term choroido-retinitis is therefore preferable.

The truly inflammatory forms of choroiditis include disseminated choroiditis, diffuse choroiditis, anterior choroiditis, purulent choroiditis, and tubercle of the choroid.

**Disseminated choroiditis** is the commonest form, and is generally syphilitic in origin. In the early stages round yellowish spots, with ill-defined edges, are seen scattered about the fundus. They are due to exudation in the choroid. The overlying retina is oedematous and cloudy. There may be dusty vitreous opacities. The membrane of Bruch becomes absorbed over the spots and the exudate becomes organized, fusing the retina and choroid into a scar. During this process the retinal pigment epithelium becomes broken down and the pigment is heaped up at the edge of the scar; other patches of pigment may become entangled in the scar tissue. In the later stages, therefore, the spots look white, and usually have a rim of dense black pigment around them, and often irregular spots of pigment within them. These spots are very sharply defined, and remain permanently. This cicatricial stage is naturally much more commonly observed than the transient acute stage. Fresh foci, however, frequently arise and pass through the same stages, until the whole fundus, or large portions of it, become covered with the atrophic spots. In the severest cases the vitreous opacities increase and the lens may become cataractous.

Vision is impaired by the destruction of the retina in the affected spots and by vitreous opacities. If the spots are peripheral, the minute scotoma does not attract attention, and extensive disseminated choroiditis may exist without very marked obscuration of vision. If, however, a spot occurs in the macular region or near it, the exudation in the acute stage separates the neuro-epithelial cells, crowding up those around, thus leading to distortion of objects (*metamorphopsia*). Separation of the cones causes objects to look small (*micropsia*); straight lines often appear bent. Flashes of light (*photopsia*) may also be experienced owing to irritation of the neuro-epithelium. There may, too, be black spots in front of the eyes, due to positive scotomata. In the later stages the atrophic spots are incapable of originating nerve impulses, and only negative scotomata exist. Although often

widely scattered, these do not usually affect peripheral vision sufficiently to prevent the patient from getting about, but a central scotoma of this nature will prevent reading and fine near work.

Though choroiditis is generally due to syphilis, either acquired or congenital, other cases occur and are probably due to auto-intoxication from some source of sepsis (e.g. pyorrhœa alveolaris).

**Treatment.**—The syphilitic cases must be treated by antisyphilitic measures. In other cases a source of sepsis must be sought for and, if found, eliminated. When no cause can be found, treatment by the methods advised for iridocyclitis is indicated. All near work should be stopped and dark glasses worn.

**Diffuse choroiditis**, though sometimes syphilitic, is probably more often due to septic or other toxins, e.g. tubercle. In the acute stage yellowish-white or grey areas are seen in the fundus, shading off indefinitely at the edges. The plaques spread and coalesce. The exudates organize, forming white areas in which the characteristically anastomosing choroidal vessels are seen, and over which the retinal vessels pass. Irregular patches of black retinal pigment appear in and at the edges of the patches. Islands of normal fundus, often sharply defined, are left between the patches. Owing to the large areas involved, vision is very seriously impaired.

**Anterior choroiditis** is usually syphilitic. It is essentially disseminated choroiditis confined to the periphery of the fundus. Hence it causes few symptoms, and may be discovered accidentally with the ophthalmoscope. It is of some value as evidence of syphilis, but similar changes are sometimes found in myopic eyes.

**Purulent choroiditis** may be exogenous, due to a perforating wound, or endogenous. The exogenous form usually passes on to panophthalmitis. Purulent choroiditis of the endogenous form has in certain cases been proved to be due to various organisms, e.g. pneumococci, staphylococci (in furunculosis), meningococci (in pseudoglioma), etc. Even when the choroid is chiefly affected, other parts of the uveal tract are usually involved, and cyclitis or hypopyon may be present. Ophthalmoscopically the media are hazy, so that the yellow oedematous retina is only dimly seen. In milder cases there is a definite focus in the fundus, usually resembling conglomerate tubercle in appearance—i.e. there is an area occupied by a cloudy-white or yellowish exudate. Owing

## UVEAL TRACT, AFFECTIONS OF

to the attenuation of the virulence of organisms which have reached the eye by the bloodstream, the process may stop spontaneously, absorption and organization of the exudates take place, and the resulting defect of vision depends on the size and position of the area involved. It is probable that many of the cases of choroiditis once attributed to syphilis or tubercle are of septic origin, and this should be carefully borne in mind in searching for the cause and treating individual cases.

**Degenerative forms of choroiditis** include those secondary to the inflammatory forms, such as the atrophic patches of disseminated choroiditis, etc., and those which are primary. Degeneration of the vessels of the choroid leads to migration of the retinal pigment, as is seen in congenital and syphilitic retinitis pigmentosa (see **RETINA, AFFECTIONS OF**). Localized patches of primary choroidal atrophy occur, and are usually central or circumpapillary.

**Central choroiditis** or, better, **choroïdal atrophy** is found in myopia, in syphilis, as the result of a blow on the eye, and in old age. In the traumatic cases the blow is followed by pigmentation in and around the fovea, resembling central senile choroidal atrophy (see below). No changes may be found immediately after the blow, and central vision may be fairly good, yet the vision gradually dwindles and the pigmentary changes appear.

**Central senile choroïd atrophy** is not uncommon in old people. If central vision is bad in an elderly patient, cannot be improved with glasses, and no cause such as cataract, glaucoma, etc., can be found, the pupil should be dilated with cocaine, or homatropine and cocaine, and the macula examined. (Care should be taken to instil eserine after the examination, on account of the risk of setting up glaucoma.) Pathological changes will then often be found. In the early stage there is a ring of fine pigment spots around the fovea, more sharply defined on the foveal side, which has a circular or wavy edge. The stippling dwindles off peripherally. The centre gradually becomes paler and the stippling denser. Frequently the pigment spots are rather larger, and irregularly scattered over the foveal region, the intervening spaces being yellowish-red and paler than the surrounding fundus. Central vision is much impaired, and eventually the central scotoma becomes absolute, preventing all reading and fine near work. The process is very slow, and one eye usually precedes the other by some weeks or months,

## UVULA, ELONGATION OF

but eventually both succumb. The condition has been attributed on insufficient grounds to sepsis, especially pyorrhœa alveolaris.

**Central areolar choroïdal atrophy** is a large central patch of choroidal atrophy also occurring in old people. The patch is oval or circular, and includes the area between the disc and the macula. The sclerotic shines through the atrophic choroid, of which all except a few of the larger vessels have disappeared. The patch is white and usually sharply defined, without much pigmentation. There is a large absolute scotoma.

**Central guttate choroiditis**, or Tay's choroiditis, is a condition found in old people in which the macular area is studded with minute yellowish-white spots. They increase in numbers, but otherwise remain stationary. The smallest are round, the larger, always small, have crenated edges due to fusion of contiguous spots. The edges of the spots are often greyish, owing to pigment epithelium which is stretched over them. The condition is bilateral, and does not in itself cause much impairment of vision. The spots are hyalin excrescences (colloid bodies) on the membrane of Bruch.

**Circumpapillary choroidal atrophy** is found in chronic glaucoma, old age, and myopia. The disc is surrounded by a yellowish ring, varying in breadth. Apart from the fact that the blind spot is enlarged, vision is not affected by this condition alone.

**Myopic choroiditis** is described under Myopia (see **REFRACTION AND ACCOMMODATION, ERRORS OF**), and **sarcoma of the choroid** under **RETINA, AFFECTIONS OF** (p. 115).

J. HERBERT PARSONS.

**UVULA, ELONGATION OF.**—Elongation of the uvula generally occurs in the presence of sinus suppuration or nasal obstruction, but some cases are due to excess in drinking or smoking. Congenital elongation is met with occasionally. The usual symptoms are an irritating cough and retching. The nasal disease must be treated, and most cases will then recover, if the organ be painted with glycerin of tannic acid or Mendl's solution (iodi puri 6 gr., potassii iodidi 20 gr., ol. menthæ piperitæ 5 min., glycerin 1 oz.) daily for a few weeks. In some cases the uvula needs removal. Having been painted with a 10-per-cent. solution of cocaine, it is seized with a pair of toothed forceps and drawn forwards; as much of the organ as is necessary can then be cut off with scissors.

G. N. BIGGS.



## VACCINATION AND VACCINIA

**VACCINATION AND VACCINIA.**—Vaccination is a method by which vaccinia, or cowpox, is inoculated into the human subject as a protection against smallpox.

**Historical note.**—Prior to the introduction of vaccination, inoculation of smallpox, or variolation, was practised in the eighteenth century, having been introduced into England by Lady Mary Wortley Montagu from Constantinople in 1721. The inoculated disease was usually of a mild type, though fatal cases were not infrequent; but the chief objection to the practice was the fact that it always gave rise to new foci of infection. In 1796 Edward Jenner performed his first vaccination, and two years later published his "Inquiry into the Causes and Effects of the Variolæ Vaccinæ," in which he showed that cowpox, casually communicated to man, has the power of rendering him insusceptible to smallpox.

During the earlier part of the nineteenth century vaccination rapidly took the place of inoculation. It was provided gratuitously by the first Vaccination Act of 1840, when variolation was made illegal, and it became compulsory in 1854. Exemption on the ground of conscientious objection was granted in 1898, when the age for obligatory vaccination was raised from 3 months to 6 months, and in 1907 the means for obtaining exemption were facilitated. Revaccination is not compulsory in this country.

**Technique of vaccination.**—The most convenient site is the skin of the left arm, about the insertion of the deltoid. The skin is first washed with soap and water, then with alcohol, and dried. Antiseptics are not required, and if used should be washed off with water before the operation, as otherwise they may interfere with the action of the vaccine. The skin is scraped with a blunt lancet or needle, and the lymph is rubbed into the scarified areas. By the official regulations four vesicles should be produced, and the total area of the vesicle-formation should not be less than half a square inch.

The use of calf-lymph has now almost entirely superseded that of human lymph, its advantages being that an unlimited supply of it is always available, and that by this method there is no danger of the transmission of certain diseases, especially syphilis, as sometimes occurred after vaccination with human lymph.

The lymph is obtained by vaccinating a young calf which has first been kept under observation for a week to exclude the possibility of tuberculosis, glanders, foot-and-mouth disease, tetanus, and skin eruptions of any kind. The vaccine virus is usually taken from the animal between the fifth and eighth days. Glycerin, 60 per cent., is added; this exercises a germicidal action without interfering with the activity of the vaccine virus.

The period at which vaccination should be performed varies according to circumstances. Unless the child is exposed to smallpox (as when the mother is suffering from that disease), vaccination should not be performed until at least ten days after birth, as until then there is a danger of hæmorrhagic disease of the new-born being set up by the operation. Vaccination is also contraindicated when the child is suffering from a generalized skin eruption or congenital debility. In the absence of such contraindications the child should be vaccinated at some time within the first three months of life.

Revaccination should be practised whenever there is a likelihood of fresh exposure to smallpox. The average period of complete immunity conferred by primary vaccination is seven years, but it is well to err on the safe side by revaccinating any persons during a smallpox epidemic whose last vaccination dates from more than five years previously. In the absence of any such epidemic, revaccination may be delayed until the age of 9 or 10 is reached. The protection afforded by successful revaccination is usually more prolonged than that of primary vaccination, but it is always advisable to repeat the operation if there is an epidemic of smallpox.

A person should not be regarded as insusceptible to vaccinia until the process has been repeated several times with different varieties of lymph, failure being often due to an old and inactive virus. Persons refractory to vaccinia are not necessarily immune to smallpox, though it is probable that if revaccination has been performed two or three times unsuccessfully with a reliable lymph there is no risk of contracting variola.

**Prophylactic efficacy of vaccination.**—The most valid arguments in favour of vaccination are these:

1. Prior to the general introduction of vac-

## VACCINATION AND VACCINIA

cination in infancy smallpox was pre-eminently a disease of children, just as measles is to-day. According to Bernouilli fully two-thirds of all children born were sooner or later attacked by smallpox, and on an average one-twelfth of all children succumbed to the disease. The protection afforded by infantile vaccination has been to raise considerably the age-incidence of smallpox, so that the great majority of vaccinated patients in a smallpox hospital is always found to be adults.

2. The low incidence and mild character of the disease in children who have been vaccinated, as compared with the unvaccinated. Thus, during the London epidemic of 1901-2, of 963 smallpox patients in the first seven years of life, only 47 had been vaccinated, all of whom recovered, while of the 916 unvaccinated cases 393 died—a mortality of 42.9 per cent.

3. The immunity enjoyed by the medical, nursing, and domestic staff of smallpox hospitals, in whom vaccination has been carried out.

4. The comparative fatality from smallpox among the vaccinated and the unvaccinated. During the London epidemic of 1901-2 the mortality among 6,945 vaccinated cases was 11.5 per cent., as compared with 33.1 per cent. among 2,278 unvaccinated cases.

5. The immunity from smallpox among the recently vaccinated or revaccinated. This was exemplified in the case of the well-vaccinated German Army in the Franco-Prussian War in 1870, in contrast with the ill-vaccinated French soldiers, 23,000 of whom died from smallpox, and more recently during the epidemic of 1901-2, in Glasgow, where the smallpox cases were confined absolutely to the not recently revaccinated section of the population.

A striking example of the impunity with which a hospital staff can be brought into contact with smallpox is supplied by the last great London epidemic. Of 974 persons employed at the Metropolitan Asylums Board smallpox hospitals during 1902, only 2 contracted smallpox. One of these was a nurse who had not been revaccinated, as she had had an attack of smallpox five months previously. The other patient had a very mild attack, and the evidence of successful revaccination was doubtful. On the other hand, of 4,339 persons employed at the M.A.B. fever hospitals during the year, 196, or 4.5 per cent., contracted scarlet fever or diphtheria and 1 died.

Opponents of vaccination have urged that the decline in smallpox is due to improved sanitation and not to the protection afforded by vaccination. They ignore the fact, however, that other infectious diseases, such as measles, scarlet fever, and whooping-cough, have not shown the same decrease, but are just as prevalent as before.

**Nature of vaccinia.**—Copeman's experiments have shown that vaccinia is merely smallpox modified by transmission through several calves. A histological confirmation of this conclusion has been furnished by Councilman and others, who obtained identical results from the inoculation of vaccine and of smallpox lymph on the cornea of the calf and rabbit, the *Cytoryctes variolæ* being found in the lesions. Vaccinia, as stated in the article SMALLPOX (q.v.), is apparently due to the asexual phase in the life of this parasite.

**Symptomatology.**—The clinical course of vaccinia in man shows a remarkable uniformity. After an incubation period of about three days, in which there are no obvious symptoms apart from a slight traumatic reaction of varying degree and duration, a pale flat papule appears at the vaccination site and gradually increases in size. On the fifth day the papule becomes vesicular at the periphery, the fluid contained in the vesicle being at first thin and transparent. At the same time a reddish areola appears round the vesicle and increases in extent until the eleventh day. On the eighth or ninth day the vesicle reaches its maturity, being well filled with a turbid fluid at the periphery and slightly umbilicated in the centre. Desiccation of the pocks begins about the eleventh day. Between the fourteenth and twentieth days the scab falls off, leaving a pigmented scar which in course of time becomes pale and pitted. According to the degree of reaction the skin surrounding the pocks becomes more or less indurated and tender, and the axillary glands become inflamed.

The general symptoms consist in a slight degree of fever and malaise, headache and backache, usually more pronounced in adults than in children. The fever, as a rule, subsides rapidly and the other symptoms soon disappear. The blood shows a leucocytosis on the third day, when the papule first appears, and later when the pocks reach maturity. During the period of maturation of the pocks a generalized transient blotchy erythematous rash is not infrequently seen, especially in children.

## VACCINATION AND VACCINIA

**Revaccination.**—The phenomena of revaccination vary according to the individual and the date at which the operation is performed after primary vaccination. The symptoms may be exactly the same as in primary vaccination, showing that the immunity has been exhausted, but more frequently they are more or less modified. The incubation period is shortened and the specific reaction appears very rapidly. The papule is very soon followed by the vesicle, and the inflammation surrounding the vesicle is not so pronounced as in primary vaccination. Constitutional symptoms are often absent altogether, but are frequently as severe as, or more severe than, after primary vaccination.

**Generalized vaccinia.**—Instead of the pocks being confined to the site of inoculation, the eruption may become generalized, either as the result of auto-inoculation or from the virus having been introduced through the digestive, circulatory, or respiratory system, instead of through the skin. The time of appearance of generalized vaccinia is usually between the fourth and tenth days. The eruption is accompanied by fever and may resemble smallpox, but is distinguished from it by the absence of a prodromal stage, and by the distribution of the lesions, which are most numerous on the trunk. Generalized vaccinia is extremely rare, only 6-8 cases having been found by Chauveau among 500,000-600,000 vaccinated persons.

**Accidental vaccinia.**—It is not very uncommon for persons in charge of recently vaccinated children to inoculate themselves accidentally with the lymph from the child's arm. The face, lips, or eye are the sites most frequently affected, but several cases are on record in which the mouth, throat, and vulva have also been the sites of accidental vaccinia. Very serious results are likely to occur when unvaccinated children suffering from a skin disease such as eczema are accidentally inoculated in this way. Of 20 such cases collected by Blochmann, 6 died, and 1 completely lost the sight of the right eye. The reasons for the unusual severity of accidental vaccinia in eczematous children are: (1) The patients are not protected by previous vaccination. (2) The humanized lymph with which they are inoculated is more virulent than calf-lymph. (3) The lymph inoculated into the eczematous area reaches the circulation more quickly than when the insertion is made in the ordinary way through the healthy skin.

**Vaccinia and other diseases.**—The possibility of vaccinia lighting up latent diseases should always be borne in mind. It is doubtless through ignorance of this fact that certain diseases, especially syphilis and tuberculosis, have sometimes been wrongly attributed to vaccination. Vaccination of persons suffering from the acute exanthemata or other infectious diseases has been carried out on an extensive scale by several observers without any injurious effects. During a smallpox epidemic at Naples, which lasted several years, more than 4,000 patients in a fever hospital, suffering from various contagious diseases, were vaccinated without a single bad result. The disease pursued its ordinary course, complications were not more frequent, and the mortality was even lower than in previous years. It was also found that the various infectious diseases had no effect on the development of the vaccine-pocks (Montefusco).

**Complications of vaccination.**—Generally speaking, complications after vaccination performed with calf-lymph are very uncommon nowadays, and can only be attributed to secondary infection, which can be avoided with proper care. Neglect of proper precautions exposes the vaccinated subject, as in the case of any other wound, to various infections, such as erysipelas, tetanus, furunculosis, or impetigo contagiosa.

Although syphilis cannot be transmitted by calf-lymph as it was by human lymph, it is conceivable that performance of the operation with contaminated instruments might transmit the disease, or that the wound might be infected later.

**Post-vaccinal eruptions** are occasionally seen, but are not frequent. They were found in 36 cases, or 3.3 per cent. of 1,070 cases of vaccination observed by de Biehler, the rashes most frequently seen being erythema multiforme, scarlatiniforme, morbilliforme, and urticaria.

**Mortality.**—The deaths certified yearly as attributable to "cowpox and other effects of vaccination" have been shown by Acland to be seven times fewer than those due to chloroform, and even fewer than those due directly or indirectly to ether. J. D. ROLLESTON.

**VACCINE-THERAPY** (see IMMUNITY).

**VACCINIA** (see VACCINATION AND VACCINIA).

**VAGABOND'S DISEASE** (see PIGMENTATION).

## VAGINA, CYSTS OF

**VAGINA, CYSTS OF.**—Practically all true cysts of the vagina arise in embryonic vestiges, either Wolffian or Müllerian. Cystic swellings are, however, encountered which have originated as myxomatous degenerations in mesoblastic tumours or as hæmatomata.

The Wolffian cysts are found on the anterior and antero-lateral surfaces of the vagina. Commonly there is one main cyst, and connected with it, above or below, a chain of small cysts, occupying a line in the long axis of the canal. They arise from that portion of the Wolffian body (Gärtner's duct) which originally ran down each side of the uterus to the base of the bladder. These cysts are thin-walled in proportion to their size, which rarely exceeds that of a cricket-ball; they have clear watery contents, and microscopically are found to be lined with cylindrical epithelium, which is flattened to cuboidal in the larger swellings.

The Müllerian cysts are commonly found in the vaginal vault. They are really misplaced glands of the uterine cervix, seldom attain any size, and contain mucoid fluid; they are lined with columnar mucous epithelium.

Vaginal cysts are frequently discovered accidentally on examination for some other condition, or during labour. Sometimes advice is sought for what is supposed to be a cystocele or prolapse owing to a mass presenting at the vulva. They seldom attain any size, and are usually sessile and glistening-white in appearance. They are readily diagnosed from a cystocele or a prolapse on digital examination, and from a cystic fibroma by the appearance and irregular density of the latter.

**Treatment** consists in enucleation. If they are at all large, it is necessary to evacuate the contents, in order to obtain a clear field of operation. Occasionally the vaginal cyst may extend into a broad-ligament cyst.

Tapping and injection of fluids is not advised, as the convalescence is more prolonged and abscesses occur. The results do not compare favourably with those obtained by enucleation.

BRYDEN GLENDINING.

## VAGINA, MALFORMATIONS OF (see AMENORRHEA).

**VAGINA, NEW GROWTHS OF.**—The vagina is seldom the seat of new growth. A fibroid tumour and malignant disease, which is usually secondary, are those found.

The **vaginal fibroid** is more allied to a fibroma than to a fibromyoma. It is usually single, sessile, of dense hardness, spherical,

## VAGINA, NEW GROWTHS OF

rarely nodular, and subject to myxomatous degeneration.

**Symptoms, diagnosis, treatment.**—The symptoms arise either from pressure effects on the bladder, urethra, rectum, or sacrum, from presentation at the vulva, or from obstruction of labour and coitus. The smaller tumours are readily diagnosed by determining their site of origin, but digital exploration may be difficult with a large tumour distending the vagina. The treatment consists in enucleation. With large tumours a portion may require to be removed piecemeal, in order to facilitate delivery and obtain a clear view of the base of attachment.

**Carcinoma of primary origin** is very rare, and provides not more than 1 in 5,000 of the cancers of the genital tract. It is seen in patients between 50 and 60 years of age, and has usually been attributed to a long-worn pessary. The growth is of the squamous-cell variety.

**Secondary cancer** is more common, and originates from the extension of a cervical cancer or an implantation of a columnar-celled growth of the uterine body.

**Chorion - epithelioma** may be either primary or secondary. The growth is a dark, vascular, small mass, readily bleeding, and appears to consist of nothing more than blood-clot. The antecedent gestation should lead to the recognition of the type of malignancy.

**Sarcomata** occur either as (a) melanosarcomata arising near the outlet, very malignant, and characterized by their dark colour; (b) tumours of a mixed solid type—myxo-sarcomata and fibro-sarcomata; and (c) polypoid growths in relation to the cervix. Of the polypoid growths, over 80 per cent. occur in children.

**Symptoms and treatment.**—The symptoms of malignant disease, absent in the early stages, are a bloodstained discharge, a tumour, and a variable amount of pain. All malignant growths should be widely excised. In inoperable cases irradiation with X-rays or radium may ameliorate the symptoms.

BRYDEN GLENDINING.

## VAGINA, PROLAPSE OF (see PELVIC ORGANS, FEMALE, DISPLACEMENTS OF).

**VAGINAL AND UTERINE FISTULÆ.**—Under this head will be considered urinary, fæcal, and menstrual fistulæ.

### 1. URINARY FISTULÆ

Impacted labour and instrumental deliveries are still responsible for the majority of urinary fistulæ. But with the more frequent recourse to Cæsarean section in difficult labour there has

## VAGINAL AND UTERINE FISTULÆ

of late years been a great decrease of such sequelæ. Operations, such as vaginal Cæsarean section, symphysiotomy, and, more especially, the wide pelvic resections for carcinoma of the cervix, account for a large proportion of the uterine group of fistulæ.

**Types of urinary fistulæ.**—The commonest type is the *vesico-vaginal* fistula, characterized by continual incontinence while the patient is up and about, while when she is recumbent it is only intermittent, as the vagina fills until movement causes escape. Confirmation of the position and type is obtained by examining with a speculum while coloured fluid, run through a catheter into the bladder, is watched as it escapes by the fistulous opening.

A rare form is the *urethro-vaginal* fistula, in which urine escapes during the act of micturition only.

In the *vesico-cervical* fistula, which is very rare, the coloured fluid injected into the bladder escapes from the cervix.

The *uretero-vaginal* fistula—the second in order of frequency—is characterized by incontinence, while the urine from the opposite side is voided naturally. The diagnosis is made by the position of the fistula in the vaginal vault and by the cystoscopic catheterization of the affected ureter to the point of the fistulous opening, when a few drops of collargol may be run through the catheter to confirm.

In the *uretero-cervical* fistula—a very rare form—the diagnosis is made as for uretero-vaginal, except that the escape, in this instance, is from the cervix.

**Symptoms.**—The symptoms of urinary fistula are the escape of urine, an unpleasant ammoniacal odour, erythema of the external parts, and a sodden vagina. Formerly these patients led a most miserable existence, but to-day the only question to be answered is, how long to wait before operating. In the presence of sloughs, pus-bearing granular surfaces, and cystitis, it is advisable to adopt palliative measures, directed towards clearing up the cystitis, obtaining healthy wound surfaces, and preventing excoriation by the application of ointments, for a period varying from three to six months. Spontaneous healing occurs, but must be rare, and is never recorded after the first month.

**Treatment** is invariably surgical, and success depends largely on special experience.

### 2. FÆCAL FISTULÆ

Fæcal fistulæ result most commonly from the unskilled use of forceps, which usually gives rise to an ano-vaginal fistula; from malignant growths; from the wearing of pessaries; and from surgical treatment of pelvic abscesses and tumours. They are characterized by incontinence of fæces and the escape of flatus.

In the commonest form a ruptured perineum fails to heal and an *ano-vaginal opening* is present, situated about an inch above the anus, with only a bridge of tissue separating it from the perineum.

The *recto-vaginal* type may occupy any part of the postvaginal wall and fornix.

**Treatment.**—The majority of these fistulæ tend to close spontaneously, and operative measures should therefore be postponed for two to four months, in anticipation of such a result and the subsidence of tissue infection.

In the *ano-vaginal* type it is usually advisable to cut the bridge of perineal tissue and then to proceed to remove scar tissue, dissecting free the anal and vaginal walls. Using catgut, the gut-wall should first be sewn up, next the perineal body reconstituted, and finally the posterior vaginal wall sutured. Healing is common, but in severe cases, in which the anal sphincter has been much damaged and scarred, full control may not be regained.

For the *recto-vaginal* fistula a free resection of the opening with its scarred neighbourhood, separation of the respective walls, and suturing in layers is uniformly successful.

For high recto- or entero-vaginal fistulæ it is simpler and more satisfactory to operate from the abdomen.

### 3. MENSTRUAL FISTULÆ

These fistulæ result from a Cæsarean section in which the uterus has been drained or sup-puration has occurred, but they also follow operations on the uterus.

A fistulous track passing from the anterior abdominal scar into the uterus permits a continual escape of clear fluid or pus, and of a periodic bloodstained discharge corresponding with the onset of the menses.

Spontaneous closure is the rule. When the fistula persists, excision of the sinus with careful suturing of the uterine and abdominal walls is uniformly successful.

BRYDEN GLENDING.

## VAGINITIS

**VAGINAL PESSARIES** (see PELVIC ORGANS, FEMALE, DISPLACEMENTS OF).

**VAGINISMUS** (see DYSpareunia).

**VAGINITIS.**—Inflammation of the vagina from infection or irritation.

**Etiology.**—The vagina is not easily infected, or damaged by chemical or mechanical irritation, because of the thickness and resistance of its mucous membrane. The acid secretion which moistens the vagina has germicidal properties, but when the discharge is abnormal it usually becomes alkaline, and the vagina in consequence is less protected from infection.

Primary vaginitis of infective origin is rare in adults, but in infants and young girls, in whom the mucous membrane is more delicate, infective vulvo-vaginitis is common and readily transmitted; though it is often due to the gonococcus, other organisms and intestinal parasites are responsible for many cases.

While infection is generally the cause of vaginitis, in the majority of these cases its primary seat is the cervix, the discharge escaping into the vagina and causing inflammation either by the organisms infecting the mucous membrane or irritating it by their products. The gonococcus and streptococcus are the two most important, but others, such as the colon and diphtheria bacilli and various fungi, are also responsible.

Douches used too hot, too strong, or too often are other causes; and just as the skin may be irritated by certain solutions, so may the vaginal wall.

Caustics applied to the cervix may run down and cause vaginitis; or the condition may result from neglected pessaries, foreign bodies, sloughing fibroids, or cervical carcinoma.

**Symptomatology.**—The most constant sign of vaginitis is discharge, but other signs and symptoms may be present, varying with the cause of the inflammation, the duration of the attack, and the condition of the mucous membrane.

If the inflammation is *acute*, irritation of the vulva is usually noticed first, and is followed by burning and throbbing at the vaginal orifice, with scalding on micturition. Within a day or two of the onset of symptoms there is usually abundant discharge, which may be thick and purulent or of a serous or sero-purulent character. On inspection, the vaginal walls and the inner surface of the labia are found to be engorged, hot, and tender, and, on wiping the mucous membrane, inflamed and bleeding

papillæ may be seen. The labia may also be œdematous.

In *chronic* cases the patient complains of discharge, tenderness of the vagina, and often of irritation of the vulva. Backache and impaired health are common, but in many cases the only symptom is discharge. When the vaginal wall is inspected, reddened areas may be found scattered over its surface. The discharge varies in amount and may be yellow, white, or greenish.

*Senile* vaginitis occurs after the menopause, and is a result of postclimacteric atrophy of the mucous membrane, which lowers its resistance to infection. There is usually free discharge; the mucous membrane is smooth, pale, and atrophied, with large red areas scattered over it; the superficial epithelium is shed in places, and the raw surfaces may adhere to one another. In this way the fornices may become obliterated or the vagina be narrowed and distorted.

In *granular* vaginitis the mucous membrane is roughened by numerous nodules; it is probably of gonorrhœal origin, and is usually associated with pregnancy.

*Membranous* vaginitis, in which a membrane forms on the wall, may occur in diphtheria, cholera, dysentery, and scarlatina. Thrush sometimes develops in this situation. Membranous vaginitis of unknown origin occasionally occurs in association with a similar condition in the intestine.

**Diagnosis.**—Vaginal discharge is generally of cervical or uterine origin; therefore the mere presence of discharge must not be regarded as evidence of vaginitis. If the vaginal mucous membrane is inflamed and papillæ, red areas, or granules are found, a diagnosis of vaginitis can be made.

**Prognosis.**—The prognosis is usually good, but in gonorrhœal cases recovery may be extremely slow because of the difficulty of eradicating the infection from the cervix and uterus; when senile change or carcinoma is the cause, relief by douching is all that can be expected.

**Preventive treatment.**—To avoid vaginitis in infants, mothers should be instructed to keep the child's vulva clean; they should also be warned of the possibilities of infection being carried to others by sponges or towels.

In adults, douching as a routine practice should be discouraged and, when douches are indicated, their frequency, temperature, nature, and strength should be regulated. Pessaries

## VAGINITIS

must not be too large; they should be kept clean and renewed three or four times a year. Sexual intercourse should be restricted within moderate limits, and, with gonorrhoea in either sex, should be forbidden until all evidence of it has been absent for at least six months.

**Remedial treatment. Vulvo-vaginitis in children.**—At the onset of the attack, search should be made for its cause, and the possibilities of a foreign body in the vagina should not be overlooked; examination for gonococci and other organisms is most important.

The child should be confined to bed and the vulva frequently bathed with weak potassium permanganate or with boric-acid solution. Precautions should be taken, especially in gonorrhoeal cases, to prevent the patient from carrying the infection to the eyes. If the inflammation does not subside the vagina can be irrigated from above downwards, or solutions may be applied to the lower part of the vagina on cotton-wool or introduced through a catheter in small quantities. Useful preparations for gonorrhoea are silver nitrate 2 per cent., argyrol 4 per cent., and colloidal silver.

**Acute vaginitis.**—In treating acute vaginitis, the patient should be confined to bed, a purge given, and light diet ordered. Some of the discharge should be collected in a sterile tube, and smears made from it for bacteriological examination.

Irritation and pain at the vulva usually cause great discomfort and require treatment. Douches of normal saline, or boric-acid or lead lotion, should be used to wash away the discharge and relieve pain and irritation. Fomentations applied to the vulva, and olive oil or paraffin smeared over the vaginal walls and vulva, sometimes give great relief.

Strong antiseptic douches increase irritation and should never be used.

In gonorrhoeal cases it is probable that the cervical glands are infected and that the discharge from them is the cause of the vaginitis; treatment should therefore aim at destroying the organisms in the cervix. A speculum should be introduced daily, the os exposed, and the solution selected introduced on a Playfair's probe into the cervical canal, care being taken not to pass the probe beyond the internal os; the infection frequently remains limited to the cervix, and therefore risk of causing its spread upwards should be avoided. The best results are obtained in these cases

by using silver nitrate (10 per cent.) or one of the organic silver compounds.

When acute vaginitis is due to the streptococcus the same general treatment is indicated, but iodine or carbolic acid should be substituted for silver salts. Infection of the cervix may be the cause of the vaginitis, but in a much smaller percentage of cases than in gonorrhoea. For this reason swabbing the cervical canal is less important, and there is not the same tendency for the infection to become chronic if it is not done. In other cases rest and frequent douching with non-irritating fluids are the most satisfactory method of treatment.

**Chronic vaginitis** should be treated with douches; those advised for acute vaginitis, and others such as alum, zinc sulphate, and lysol solutions, may be tried. The vagina should also be treated locally by swabbing, plugs, or medicated pessaries, and the treatment of any discharge from the os should not be omitted.

**Swabbing.**—The vaginal walls should be exposed through a speculum, the secretion wiped away, and silver nitrate 2 per cent., iodine 2 per cent., or some organic silver preparation applied to the whole mucous membrane. The process should be repeated two or three times a week.

If small inflamed areas are seen, pure carbolic or stronger solutions of iodine or silver nitrate can be applied to them.

**Plugging.**—The object is, by distending the vagina, to open up all the folds in the mucous membrane and so expose its whole surface to the drug. An anæsthetic should be given if strong solutions are used. After all discharges have been wiped away, the vagina should be swabbed carefully with 5- or 10-per-cent. silver nitrate and packed with a plug moistened in this solution. The plug should be removed after twenty-four hours, and normal saline douches ordered three times a day for the following week. Dichloramine-T (5 per cent.) in chlorosolane, and eucalyptus oil (40 per cent.) in olive oil, give good results. An anæsthetic is unnecessary.

**Medicated pessaries** are made containing various substances such as ichthyol, iodine, colloidal silver, and thymol, in a medium that melts at body temperature. These pessaries are introduced into the vagina and, as they melt, liberate their active ingredients; in this way the mucous membrane is exposed to the action of the antiseptic for a long period.

A simple douche should be given the evening

## VALVULAR DISEASE, CHRONIC

before introducing the pessary, which should remain in position during the night and be followed by another douche. A course of ten days or a fortnight of this treatment often has excellent results.

J. P. HEDLEY.

### VALVULAR DISEASE, ACUTE (*see* ENDOCARDITIS, ACUTE).

**VALVULAR DISEASE, CHRONIC.**—In heart disease generally the question of *prognosis* is of the first importance to both medical attendant and patient.

The latter will want to know, not only if he is in danger of sudden death, but what are his prospects in regard to the future, and how he may regulate his mode of life so as to give himself the best possible chance. The medical attendant is required, therefore, not merely to make a diagnosis of the exact nature of the patient's affection, but to forecast what effect it is likely to have in shortening his life or limiting his sphere of activity.

During and immediately after an attack of acute endocarditis, it is frequently very difficult, if not impossible, to estimate with any degree of accuracy to what extent the particular valve attacked, or the heart-muscle itself, has been damaged. When the dilatation from the myocarditis, which in some degree is usually associated with acute endocarditis, has subsided, and the damaged valve has become cicatrized, so that there is produced what is termed a "chronic valvular lesion," an approximate estimate can be formed of the extent of the lesion and a basis for prognosis is thus established. A slight valvular lesion may give rise to little inconvenience, but the establishment of a lesion of some severity upsets the hydraulics of the circulation and impairs the mechanism of the heart as a pump. As a result of a severe lesion certain changes in the cavities and walls of the heart take place which are, in part, an attempt by Nature to counteract the ill effects of, or compensate for, the lesion. The vascular system also adapts itself to meet the requirements of the circulation. Thus, in mitral or aortic stenosis we find the vessels, of which the radial artery may be taken as an example, contracted down and small, in proportion to the degree of narrowing of the valvular orifice affected; for it is obvious that if the vessels remained the normal size, the attenuated stream flowing into them could not maintain the pressure requisite for an efficient circulation. On the other hand, in aortic in-

competence, a condition in which a powerful hypertrophied left ventricle throws into the arterial system, with great force, an abnormal quantity of blood, we find the arteries large and relaxed to accommodate the increased amount, and the peripheral resistance diminished by a similar relaxation of the arterioles, so that the work of the heart is proportionately lessened.

**Reserve power of the heart.**—In the healthy heart there is an enormous reserve power which enables it to respond to the increased demands entailed by exertion and exercise without any ill effect. Though the most important factor in this reserve power is the efficiency and integrity of the muscular wall of the heart, it is obvious that serious damage to a valve will impair the functional efficiency of the cardiac mechanism. It is important, therefore, for purposes of prognosis, that we should be able to form some estimate of the degree of severity of a valvular lesion. How this may be arrived at will be discussed in the sections on the different valvular lesions.

### AORTIC INCOMPETENCE

**Etiology.**—The valvular lesion may be the result of acute endocarditis in childhood or early adolescence associated with rheumatism, scarlet fever, or, more rarely, other infections, the pathology of which is considered under ENDOCARDITIS, ACUTE (q.v.), or it may be due to chronic degenerative change of the valve associated with atheroma and dilatation of the aorta. It is important to distinguish between these two varieties, and, as they differ materially in their clinical features, they will be discussed under separate headings.

**Pathological anatomy.**—The condition of the valves varies according to the degree of severity of the attack of endocarditis. In *mild or simple rheumatic endocarditis*, which occurs usually in childhood or early adolescence, along the margins of the valve small raised pinkish granulations are present, which microscopically are seen to consist of a superficial layer of fibrin and leucocytes, beneath which is proliferating connective tissue. In the process of healing, fibrous tissue is formed, and, a certain amount of cicatricial contraction taking place, the margin of the valve becomes irregular and allows of a varying amount of leakage. In the *more severe and ulcerative forms of endocarditis* there is considerable destruction of tissue; if the patient survive and repair take place, the valve is so greatly deformed



## VALVULAR DISEASE, CHRONIC

and its flexibility so impaired that it allows of considerable reflux. Frequently, also, lime salts are deposited in the diseased areas and the valve becomes rigid and calcified. Its cusps may become adherent at their margins and, as cicatrization takes place, may contract down, a narrow rigid stenosed orifice resulting (aortic stenosis).

In the *chronic degenerative lesions* of later life, resulting most commonly from syphilis, but

competence there is a powerful hypertrophied left ventricle throwing into the arterial system with great force an abnormal quantity of blood, and the vessels, by relaxing, increase the arterial capacity, and accommodate the increased volume of blood thrown in at each systole. The relaxation of arterioles, by lowering the peripheral resistance, also lessens the work of the heart. The artery at the wrist is large. The pulse wave comes with a sudden rush

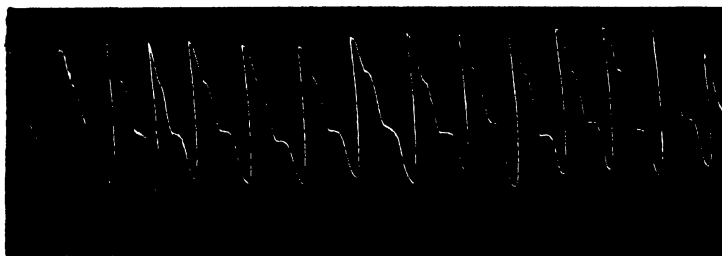


Fig. 102.—Collapsing pulse of aortic incompetence due to acute endocarditis.

sometimes from severe intermittent strain, or from tobacco or lead poisoning, the degenerative changes in the valves are usually associated with atheroma of the aorta. The valves may be rigid, sclerosed, and calcified to a varying degree. Frequently also, as a result of atheroma, the root of the aorta gives way and becomes dilated. This may be an important contributory cause of the incompetence.

**Physical signs. The pulse.**—The most remarkable feature in the pulse is its collapsing character, to which attention was first drawn

it is large and forcible, but very short and ill sustained, collapsing almost immediately under the finger. These characteristics are best brought out when the arm is raised. Another important characteristic is the position of the dicrotic notch. In a collapsing pulse it falls below the middle of the descending line in the pulse tracing to near the base, instead of to above the centre, as in the normal pulse tracing. (Fig. 102.)

In the *aortic incompetence of later life* the pulse differs materially from that just described. The vessel is usually thickened and less elas-

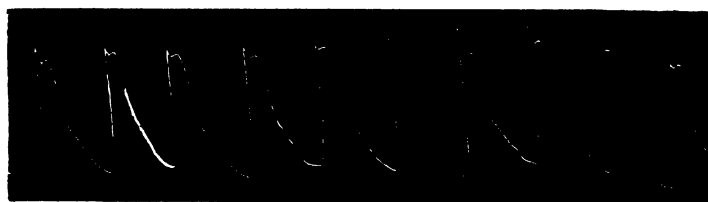


Fig. 103.—Pulse of aortic incompetence in later life due to degenerative change.

by Corrigan. It is due in part to the loss of support to the column of blood in the aorta on its elastic recoil, and consequent sudden fall of blood-pressure by leakage through the incompetent valves, and in part to the remarkable relaxation of the arteries and arterioles which is characteristic of aortic incompetence. This relaxation would seem to be a protective measure on the part of Nature. In aortic in-

competence as a result of degenerative change or high arterial tension, the mean blood-pressure is much higher, and the vessel remains more or less full between the beats. Though some features of the aortic pulse are present, the collapsing character is much less marked, the fall being more gradual, and the dicrotic wave less pronounced or absent. (Fig. 103.)

**Arterial and capillary pulsation.**—A promi-

## VALVULAR DISEASE, CHRONIC

nent feature in aortic incompetence is the violent pulsation of the arteries throughout the body, which is so conspicuous in the carotids that from this sign alone a diagnosis may sometimes be made. Capillary pulsation also is usually present, and is best brought out by rubbing the skin, preferably on the forehead, so as to bring out a red patch. This will be seen to flush and pale alternately, flushing with the systole and paling with the diastole of the heart. It is also well brought out by pressure on the finger-nail, when the pulsation is seen beneath the nail. Sometimes pulsation is transmitted through the capillaries to the veins. The capillary pulsation is the result of the general arterial relaxation previously described, which obtains in the capillaries as well as in the larger vessels.

**The heart.**—Dilatation of the left ventricle, followed by hypertrophy, usually results from aortic incompetence, and extremes of dilatation and hypertrophy are met with in this condition when a severe lesion has occurred in early life.

The dilatation appears to be brought about in the following way: during diastole, the elastic recoil of the aorta forces back a certain amount of blood through the incompetent valves into the left ventricle when it is in a relaxed and defenceless condition; at the same time blood is pouring into it from the left auricle. Thus the ventricle has either to resist the reflux, or, if it is incapable of this, accommodate an increased quantity of blood and therefore dilate. The chief factor in antagonizing over-distension or dilatation by the reflux from the aorta is the tonus of the heart-muscle; this may be greatly impaired by the myocarditis usually associated with the valvular lesion, so that dilatation results. Later on, hypertrophy follows as a result of the additional work entailed by the increased output from the enlarged ventricle.

**The diastolic murmur.**—The pathognomonic sign of aortic incompetence is a murmur produced by the backward rush of the blood from the aorta through the leaking valve into the left ventricle during diastole, as the elastic recoil of the walls of the great vessels takes place. The seat of production of the murmur being the valve at the root of the aorta, the point on the chest-wall nearest the origin of the sound is at the level of the third left costal cartilage close to the sternum, where the aortic valve is situated; but since the *conus arteriosus* of the pulmonary artery is inter-

posed between the aorta and the chest-wall, it is not always at this point that the murmur is heard best. It is usually audible over "the aortic area" in the second right intercostal space, where the aortic second sound is heard best, as the aorta comes into contact with the chest-wall at this point; but the murmur is frequently more pronounced and heard more distinctly along the left margin of the sternum in the fourth or fifth left intercostal space. This distribution of the murmur may perhaps be explained by its conduction downwards by the regurgitant stream of blood. It is also usually audible at the apex. Its seat of maximum intensity is in marked contrast to that of the murmur in aortic stenosis, which is always most audible at the aortic cartilage.

**The aortic second sound.**—In moderate degrees of incompetence this is not replaced to any extent by the murmur and is usually audible. It is not always possible, while auscultating the aortic area, to be certain that the second sound heard there is not the pulmonic conducted across; but since the aortic second sound is well heard over the carotid in the neck, a position in which the pulmonic is never audible, it is possible, in case of doubt, to ascertain, by listening there, to what extent the aortic second sound is replaced by the murmur.

**Mitral murmurs.**—As has already been pointed out, there is almost invariably some dilatation of the left ventricle as a result of aortic incompetence, and functional incompetence of the mitral orifice frequently results and gives rise to a mitral systolic murmur at the apex. The mitral valve itself may have been damaged, coincidently with the aortic valve, during the attack of acute endocarditis, and mitral incompetence may result from this also. In certain cases a presystolic murmur may be heard at the apex in the absence of any mitral stenosis, the "Austin Flint" murmur, so called because first described by Flint in 1862. An explanation suggested for its occurrence is the following: The mitral and aortic orifices being in close apposition, the aortic regurgitant stream impinges on the relaxed anterior flap of the mitral valve during diastole, and at the same time the current of blood from the left auricle strikes the opposite surface of the mitral flap, which is thus thrown into vibration and generates a murmur. An alternative explanation is that the regurgitant aortic stream prevents the complete falling back of the mitral flap, which thus partially occludes

## VALVULAR DISEASE, CHRONIC

the mitral orifice and gives rise to actual obstruction at the mitral orifice. A *presystolic murmur* audible at the apex may, however, be due to genuine mitral stenosis, and may be recognized by the characteristic rumble terminating abruptly in a short sharp first sound; but the most important factor in the diagnosis of mitral stenosis as a complication of aortic incompetence will be the modification of the pulse, which will be small in volume in proportion to the degree of stenosis, and will lose its collapsing character. Cyanosis and symptoms of pulmonary congestion and backworking may also be present, or may be induced on slight exertion if the stenosis is severe.

**Symptoms.**—The symptoms, when present, are mainly those of deficient blood supply to the systemic circulation consequent on the ill-sustained blood-pressure, to wit, pallor of countenance, shortness of breath, and a tendency to faintness from cerebral anæmia, especially on suddenly assuming the erect from the recumbent position. Sometimes præcordial pain or anginoid attacks occur. In the later stages, attacks of cardiac asthma with severe and agonizing orthopnoea may be a distressing feature.

When the left ventricle gives out and free leakage through the mitral orifice takes place, engorgement of the lungs and liver, œdema of the lower extremities, cyanosis, and a train of mitral symptoms predominates.

**Prognosis.**—For purposes of prognosis it is important, if this be possible, to form some estimate of the degree of severity of the valvular lesion. We have seen that, as a result of aortic incompetence, certain changes in the heart and circulation take place. The extent to which these changes have resulted will afford valuable help in arriving at such an estimate.

**The pulse.**—The degree of collapse in the pulse and the size of the artery are proportionate to the amount of incompetence of the aortic valves; thus, a very collapsing pulse and a large relaxed radial artery are indicative of a severe valvular lesion.

**The heart.**—The degree of hypertrophy and dilatation of the left ventricle also affords important information. As the ventricle has to propel a greater volume of blood than normal and resist, as far as possible, excessive distension by the regurgitant stream, it is essential that its walls should be strengthened. We find that in favourable circumstances hypertrophy of its walls takes place in proportion to the

increased work it has to do. If, nowever, the muscle be seriously damaged by myocarditis, or if the patient get up and go about too soon, before the heart has had time to recover from the acute attack, the degree of dilatation and hypertrophy which takes place may be disproportionate and excessive.

**The diastolic murmur and the aortic second sound.**—No special prognostic significance can be attached to the character of the murmur, which may be loud or soft, short or prolonged; but the degree to which the murmur replaces the second sound is important, and if this sound is inaudible in the neck over the right carotid artery, it is an unfavourable element in the prognosis. When the heart is failing the diastolic murmur usually becomes shorter and less pronounced.

**Symptoms.**—The readiness with which symptoms of cardiac distress are induced on exertion is one of the most important considerations in prognosis, as it is a measure of the degree to which the heart is handicapped by the valvular lesion, and also of its functional capacity. In slight cases, and even in lesions of moderate severity in which efficient compensatory hypertrophy of the left ventricle has taken place, the patient is able to live an ordinary life, to take active exercise without discomfort, and may survive for many years. In later life, as degenerative changes set in, the heart is apt to break down, and old age is seldom attained. When symptoms of insufficient blood supply to the periphery, such as pallor of face, giddiness, and faintness, are prominent, and shortness of breath is induced on the least exertion, the outlook is necessarily unfavourable.

**The aortic incompetence of later life.**—In the aortic incompetence of later life, in which it is the result of syphilis, intermittent strain, and degenerative lesions associated usually with atheroma and dilatation of the aorta, the prognosis is far less favourable than in the case of lesions due to acute endocarditis in the young. Frequently, dilatation of the root of the aorta, to which the valves are attached, is the primary lesion and the main cause of the incompetence. Moreover, from the nature of the degenerative changes which have given rise to it, the lesion is likely to progress.

If the regurgitation be at all extensive the left ventricle is unable to respond to the extra strain, or, at this period of life, to undergo hypertrophy, and consequently soon breaks down. Death may result from a sudden syn-copal attack, or secondary mitral incompetence

## VALVULAR DISEASE, CHRONIC

with backworking through the lungs and cedema of the legs may ensue.

**Treatment.**—In well-compensated or slight cases no special treatment is necessary, but instructions should be given as to the amount and kind of exercise to be taken; this will vary according to the degree of severity of the lesion.

There is some controversy as to the advisability of administering digitalis shortly after the establishment of the lesion. It has been argued that it is advantageous because it increases the tonicity of the heart-muscle, and would thus tend to limit the amount of dilatation. From experiments on animals, in which a traumatic aortic lesion has been effected, it has appeared to be of benefit in limiting the dilatation, but it does not follow that it will be useful in rheumatic lesions. In rheumatism the heart-muscle has been impaired by myocarditis, and if damaged severely, will be un-

venous system, the result of right-ventricle breakdown secondary to that of the left.

In the former case there are shortness of breath, pallor of countenance, and sleeplessness of an intractable kind, or anginal attacks; sudden death from syncope may ensue. In these circumstances, rest in bed is of course essential. Stimulants, ammonia, ether, alcohol, nux vomica, and strychnine are useful; hypodermic injections of morphia give great relief in anginal attacks.

In the second group, in which cyanosis, dropsy, enlargement of the liver, and mitral symptoms are present, digitalis is of great service, but it should not be persisted in after the mitral symptoms have been relieved, and should gradually be discontinued.

### AORTIC STENOSIS

**Etiology.**—Stenosis of the aortic orifice arises most commonly from cicatrization and



Fig. 104.—Pulse of aortic stenosis.

able to respond. Even if the muscle be so little damaged that the administration of digitalis improves its tonicity, the heart is stimulated to contract more forcibly and the tonicity of the aorta is increased, so that the reflux will become more violent. Absolute rest for some weeks or months after the establishment of the lesion till the heart has had time to hypertrophy is far safer and preferable to administering digitalis. When the patient begins to go about again, exercise should be renewed very gradually and great care taken to avoid exertion or hurry. If these precautions be observed at the outset, recovery may take place to a remarkable degree in favourable cases with a moderate lesion, and the patient may suffer so little inconvenience that he may scarcely be aware of the presence of heart disease for many years. This, of course, only holds good for lesions acquired in childhood or early adolescence.

When compensation fails and symptoms set in, they arise from one of two causes: (1) failure of the left ventricle to maintain an efficient circulation in the arterial system, (2) backward pressure and engorgement of the

adhesion of the cusps of the valves during the process of repair after an attack of acute endocarditis. It is not a very common lesion; when it occurs it is frequently associated with aortic incompetence. The degree of narrowing varies greatly; in extreme cases the cusps may become fused together to form a fibrous ring which undergoes calcification, leaving only a narrow orifice a quarter to half an inch in diameter. Degenerative changes, the result of atheroma or syphilis, may also be a cause of aortic stenosis and give rise to thickening, rigidity, and calcification of the valves, which function imperfectly and project into the bloodstream but do not cause a serious degree of obstruction. Aortic stenosis of congenital origin is occasionally met with.

**Physical signs. The pulse.**—The pulse wave is gradual in its ascent, indicating a slow filling of the aorta; the percussion wave is small, the vessel full between the beats, and the dicrotic wave ill marked or absent (Fig. 104). As stenosis is more commonly associated with incompetence, this typical pulse is not very frequently met with; but when the double aortic lesion exists, the degree to which

## VALVULAR DISEASE, CHRONIC

the pulse of aortic incompetence is modified by the stenosis, and so loses its main characteristics, is of great importance in the diagnosis.

**The heart.**—Hypertrophy of the left ventricle, induced by the effort to overcome the obstruction, is the main structural change in the heart. This may be very considerable, and as the stenosis is usually associated with aortic incompetence the heart may attain an enormous size. The aortic second sound varies in intensity, but is usually rather faint and indistinct in pronounced cases of stenosis; when, however, dilatation of the aorta is present, and the obstruction is slight and due to thickening of the valves, it may be loud and ringing.

The chief auscultatory evidence of aortic stenosis is a systolic murmur with its maximum intensity at the second right intercostal space. It is usually conducted to the carotids and to the left of the sternum. It may be soft and blowing or loud and vibratory in character. A vibratory thrill over the aortic area is felt on palpation in a certain number of cases.

It must not be concluded that every aortic systolic murmur is indicative of aortic stenosis. Such a murmur may also be caused by roughening or rigidity of the valves, dilatation of the aorta, aneurysm, or anæmia.

Hæmic murmurs can usually be identified without difficulty by the presence of anæmia and by the absence of any structural change in the heart or modification of the pulse.

Aneurysm of the ascending portion of the arch of the aorta is not difficult to distinguish when associated with a systolic murmur, for other definite signs of aneurysm—a pulsating tumour or dullness in the second right intercostal space—will be present.

In distinguishing between mere roughening or rigidity of valves and actual stenosis, it must be borne in mind that the effect of aortic stenosis is to diminish the size of the blood-stream flowing from the heart into the aorta and to throw extra work on the left ventricle. As the calibre of the arteries is reduced in proportion to the size of the blood-stream, the radial artery is relatively small in actual stenosis and the pulse modified as described above. There is also marked hypertrophy of the left ventricle. When aortic incompetence is present as well, we can judge of the degree of stenosis by the extent to which the characteristics of the collapsing pulse of aortic incompetence are modified. Mere roughening or rigidity of the valves does not cause these modifications in the pulse or changes in the heart. These

affections are usually met with in elderly subjects with a high blood-pressure and arteriosclerosis. There may therefore be a moderate degree of hypertrophy of the left ventricle, the result of high blood-pressure; the radial artery will be thickened and inelastic but not diminished in size.

**Prognosis.**—When the constriction is moderate in degree and occurs in a young subject, the heart hypertrophies sufficiently to overcome the obstruction, and no ill effects may be felt or serious symptoms arise for many years. If the stenosis be severe, the left ventricle, in spite of hypertrophy, has great difficulty in maintaining an efficient circulation. Sooner or later it wears itself out and gives way. As this takes place the pulse becomes irregular and attacks of angina or cardiac asthma may set in. Death may occur from a sudden syncope attack, or from gradual heart failure with dropsy and pulmonary engorgement.

**Treatment.**—As nothing can be done to relieve the obstruction, care must be taken not to put more strain or work on the left ventricle than is absolutely necessary, and life should be regulated carefully accordingly. When the left ventricle begins to fail and serious symptoms arise, rest in bed and the administration of digitalis in moderate doses are of service, but the results are not very satisfactory, and only a temporary improvement can be looked for.

### MITRAL INCOMPETENCE

Mitral incompetence *per se* is certainly by far the least serious of the valvular affections. The range of possibilities as regards prognosis in this affection is very wide and varied. The mere statement that a patient is suffering from mitral incompetence is absolutely valueless without qualifications, and, as will be seen later in the discussion of prognosis, the etiology of the lesion, the condition of the heart-muscle, and the degree of dilatation of the left ventricle are of great importance in this connexion.

**Etiology.**—From this point of view the varieties of mitral incompetence may be divided into two main groups: (1) incompetence due to a lesion of the mitral valves from endocarditis; (2) incompetence due to atony or dilatation of the left ventricle, the valves themselves being normal.

(1) **Mitral incompetence due to valvular lesions.**—Damage to the mitral valve from acute rheumatic endocarditis is by far the commonest cause in this group. Chronic

## VALVULAR DISEASE, CHRONIC

endocarditis due to thickening and rigidity of the mitral valve may be a cause of incompetence in later life, but this is usually of slight degree and is not serious unless associated with degenerative change in the heart muscle.

(2) **Mitral incompetence due to dilatation of the mitral orifice.**—In discussing mitral incompetence dependent upon atony or dilatation of the left ventricle, we must bear in mind that the mitral ring to which the valves are attached is not rigid and unyielding like that of the aorta, but a circular band of muscle-fibres which by their contraction in systole play an important part in the closure of the orifice. Further, the papillary muscles attached by the chordæ tendinæ to the margins of the valves are also factors in the mechanism which secures accurate apposition of the valvular curtains. They prevent them from being unduly displaced upwards by the strain to which they are subjected during the ventricular systole. If, therefore, the contractile power of this circular muscle, or that of the papillary muscles, be impaired by loss of tonicity, slight leakage through the mitral orifice may result.

When the whole left ventricle dilates and its walls give way as a result of damage to its muscle-fibres by myocarditis or degenerative changes, a proportionate degree of relaxation or enlargement of the mitral ring necessarily results, and a considerable amount of leakage may take place through the imperfectly closed mitral orifice.

Loss of tone in, or damage to, the muscular fibres of the heart may be brought about by various causes. There is usually some loss of tone, due to the effect of bacterial toxins, in the cardiac muscle after acute illnesses such as influenza, pneumonia, and enteric fever, but this does not, as a rule, give rise to incompetence of the mitral valve unless undue exertion is indulged in too soon after convalescence.

Dilatation of the left ventricle, sufficient to give rise to mitral incompetence, is frequently met with in anæmia, Graves's disease, and chronic alcoholism; in the last two instances it may be very considerable.

Mitral incompetence from dilatation of the left ventricle also occurs secondarily to aortic incompetence, and sometimes in subacute Bright's disease (parenchymatous nephritis).

Rheumatism, also, by its toxic effects on the myocardium, is one of the most important causes of cardiac dilatation, and in the relapsing rheumatic pericarditis of childhood we

meet with serious mitral regurgitation resulting therefrom. The myocardium in these cases is always affected, and frequently the pericardium becomes adherent to a greatly dilated heart, fixing it in a condition of dilatation and hampering its movements. The left ventricle may thus remain permanently enlarged and greatly dilated, so that a serious degree of mitral incompetence persists.

**Physical signs. The pulse.**—The pulse is usually regular in force and frequency till compensation breaks down; but in old rheumatic hearts and cases where chronic degenerative change in the cardiac muscle is present, irregularity of rhythm, dependent on interference with the paths of the nervous impulses, may set in.

**The heart.**—Minor degrees of incompetence due to endocarditis may exist without giving rise to any appreciable change in the heart or position of the apex beat. When the reflux is considerable, dilatation of the left auricle, followed by some hypertrophy, results, but the main work of compensation falls upon the right ventricle, which, undergoing hypertrophy, causes some displacement of the apex beat to the left. When the incompetence is due to cardiac dilatation, the degree of displacement of the apex beat naturally depends upon the degree of dilatation of the two ventricles. It is displaced outwards and downwards in varying degree.

When the left ventricle remains permanently dilated, as is frequently the case when the pericardium becomes adherent after prolonged or relapsing pericarditis, hypertrophy ensues, as it has to propel a larger quantity of blood than normal. It may be very greatly enlarged so that the apex beat is in the sixth or seventh space in the anterior or midaxillary line.

The characteristic sign of mitral incompetence is the presence of a systolic murmur heard at the apex, due to the regurgitation of blood through the imperfectly closed mitral orifice during systole of the heart. The murmur is usually conducted to the axilla and may be audible at the back to the left of the spine at the level of the fifth to the eighth ribs, where the shoulder of the left ventricle rests against the spine. The extent to which the murmur is conducted depends on its intensity and pitch. It may be soft and blowing in character or, in some instances, high-pitched and musical, and may be short or long in duration. It may replace the first sound entirely or partially, or may immediately follow without appreciably affecting it.

## VALVULAR DISEASE, CHRONIC

The pulmonary second sound is accentuated, due to increase of pressure in the pulmonary circulation.

When the right ventricle gives way and compensation fails, there are dyspnoea and cyanosis, the veins of the neck are full, and pulsation of the ventricular type may occur. The liver becomes enlarged and oedema of the lower extremities develops, but it is remarkable how rapidly both the dropsy and the enlargement of the liver will disappear under rest in bed and suitable treatment. Examination of the liver should always be carried out carefully, as its fluctuations in size afford important evidence of the degree of backworking and engorgement of the right side of the heart.

**Prognosis.** (1) *In cases due to a valvular lesion.*—When we are called upon to give a prognosis in a case of chronic valvular lesion traceable to a past attack of rheumatic endocarditis, our task is comparatively simple. We must first endeavour, by the data at our command, to estimate the degree of severity of the lesion.

The characters of the murmur and of the first sound afford important information. If the murmur be not conducted much beyond the apex beat, not audible in the back, and do not replace the first sound to any great extent, or if it follow the first sound after a brief but appreciable interval (i.e. is late systolic in time), the regurgitation is presumably slight in amount. If the systolic murmur replace entirely or greatly modify the first sound, we may infer that the lesion is severe, for the intraventricular pressure is so reduced by the regurgitation that it is insufficient to make the muscular wall, as it suddenly contracts down on the contained blood, taut enough to generate that sound.

As the work of compensation falls mainly upon the right ventricle, the degree of enlargement and hypertrophy of the right ventricle is an important guide to the severity of the lesion. Enlargement of the right ventricle causes displacement outwards and somewhat downwards of the apex beat, and epigastric pulsation. The degree of accentuation of the pulmonic second sound affords useful information as to the extent to which the pressure is increased in the pulmonary circulation.

*The symptoms.*—A very important factor in prognosis is the readiness with which symptoms of cardiac distress are induced, or a breakdown of compensation occurs. Many people are going about with a mitral lesion who are utterly

ignorant of the fact, and lead an ordinary life, taking plenty of exercise without suffering any inconvenience. In them we find little appreciable evidence of compensatory changes, and it is obvious that in such cases the lesion is so slight as to be almost negligible, whatever the character of the murmur.

In other cases the patient, though unconscious of any heart affection, seeks advice for shortness of breath on going uphill or upstairs, though comfortable when taking ordinary walking exercise. This indicates that the reserve power of the heart has been considerably reduced by the derangement caused by the valvular lesion, extra exertion inducing symptoms of cardiac distress. In these cases it is necessary to lay down strict rules as to the amount and nature of the exercise allowed, and to explain to the patient that these symptoms are a warning to desist at once from the exertion which induces them.

In regard to prognosis in cases of complete breakdown of compensation in which enlargement of the liver, dropsy, and other signs of engorgement are present, much depends upon whether the breakdown has been induced by undue exertion, or by any additional burden on the heart such as an attack of bronchitis, or has occurred while the patient has been taking all possible precautions. In the former event it is remarkable how, time and again, the heart responds to suitable treatment by rest and the administration of purgatives and digitalis. In the latter, the prognosis is necessarily unfavourable, the more so as the onset of symptoms is likely to be due to commencing degenerative change in the cardiac muscle.

A danger to be apprehended, in cases of mitral incompetence in children who come under notice shortly after an attack of endocarditis, is the subsequent onset of mitral stenosis from cicatricial contraction of the damaged valves. This should make our prognosis guarded even in apparently favourable cases. My impression is that the patient often comes up for medical advice only when mitral stenosis has set in and symptoms attributable to that affection begin to present themselves, although mitral incompetence may have been present for some years.

(2) *In cases due to dilatation of the mitral orifice.*—In dealing with the question of prognosis in mitral incompetence due to dilatation of the left ventricle, without actual lesion of the valves, the etiological factors are of extreme importance, and are many and varied.

## VALVULAR DISEASE, CHRONIC

When, during an attack of rheumatism, a systolic murmur, indicative of mitral incompetence, develops at the apex, it is often a difficult matter to decide whether this is due to damage to the valves from endocarditis or is the result of dilatation of the left ventricle. If the murmur be musical in character or change in quality while the patient is under observation, we may be fairly certain that it is the result of an actual lesion of the valves. If there be little evidence of dilatation of the ventricle, only slight displacement of the apex beat, and no great increase in the area of cardiac dullness, and the murmur be soft and blowing in character, it may be necessary to wait for some weeks after the subsidence of the attack before its nature can be determined with certainty. If, after the subsidence of an attack of rheumatic pericarditis in a child, we find that the cavities of the heart, both right and left, remain greatly dilated, and notice that there are marked symptoms of cardiac distress, such as rapid respiration and pulse, orthopnoea, and perhaps enlargement of the liver, and on auscultation at the apex we hear a loud blowing systolic murmur, we may be inclined to attribute these in part to a lesion of the mitral valve. Such a lesion may or may not be present, but is of minor importance, as in these cases the extreme dilatation and the resulting mitral incompetence are the consequence, not of a valvular lesion, but of damage to the myocardium inflicted by the toxins of the rheumatic micro-organisms. Inasmuch as the right ventricle is usually affected to the same degree as the left, and, being thinner-walled, dilates even more readily, there is no compensatory mechanism available to counteract the effects of the mitral incompetence, and, in severe or prolonged attacks, enlargement of the liver, distressing dyspnoea, and dropsy may set in. If the patient survive the attack, the pericardium frequently is universally adherent and the heart does not recover from the dilatation, but remains permanently enlarged, a loud blowing systolic murmur being audible at the apex and conducted through to the back. In such cases, even though hypertrophy follows, the prognosis as regards efficiency of compensation or prolonged life is necessarily unfavourable.

In Graves's disease and in chronic alcoholism it is remarkable how the heart may recover when the toxin responsible for the cardiac dilatation and resulting mitral incompetence has ceased to exert its influence. An illustra-

tion is a case of Graves's disease in a lady aged 40, whom I attended when the apex beat was in the sixth space in the anterior axillary line and a loud blowing systolic murmur was present; a year later, when the disease had become arrested, the apex beat had receded to the fifth space, and the systolic murmur had disappeared entirely.

Prognosis, therefore, even in severe degrees of mitral incompetence, may be favourable provided there is no serious degree of degenerative change in the cardiac muscle. *A fortiori*, it is favourable in the minor degrees of incompetence due to temporary loss of tone of the cardiac muscle from the various causes enumerated in the section on etiology.

**Treatment.**—Mitral incompetence is met with in such varied degrees and has such varied causation that it is difficult to lay down general rules as to treatment, and reference must be made to some of the different classes of cases.

In slight lesions resulting from endocarditis when no symptoms are present, no drug treatment is required, but some restrictions as to violent exercise should be enjoined. It is a mistake to give digitalis or alarm a patient unnecessarily, merely because a mitral systolic murmur is present.

In more severe lesions, in which cardiac symptoms such as shortness of breath, cyanosis, or præcordial pain are induced on slight over-exertion, strict rules as to exercise, hurrying upstairs, etc., should be laid down, and if the pulse become irregular or rapid, and shortness of breath persist, rest in bed for some days should be ordered, a purge administered, and small doses of digitalis (10–15 min. of the tincture) be given three times a day till the heart recovers its tonicity. It is important to bear in mind that patients suffering from mitral disease are especially liable to bronchitis from the congested condition of the lungs. Careful examination of the chest should always be made, so that, if the cardiac breakdown has occurred from this cause, suitable treatment by expectorants should be given previously to the administration of digitalis.

When serious symptoms, such as dropsy, cyanosis, dyspnoea associated with enlargement of the liver and pulsation of the veins in the neck, set in, steps must be taken to relieve the venous engorgement of the right side of the heart, and to improve the tonicity of the heart-muscle. To relieve the engorgement, venesection, or the application of leeches or dry cupping over the liver, may be employed



## VALVULAR DISEASE, CHRONIC

in severe cases. Venesection is especially indicated when bronchitis is present in addition. Rest in bed is, of course, imperative. Purgatives of which mercury in the form of calomel or blue pill is a constituent, combined with colocynth and hyoscyamus, and followed by salines repeated every morning, should be employed, and will often suffice to relieve the venous congestion in less severe cases. To restore tonicity and overcome the dilatation, digitalis should be given in fairly large doses (15 min. of the tincture every four or six hours); if much dropsy be present, 5-8 gr. of caffeine citrate may be added. Digitalis in the form of Nativelle's granules ( $\pm \frac{1}{10}$  gr. every four hours) sometimes proves more efficacious than the tincture, but should not be persisted in for more than three or four days, as its effects are rapidly cumulative. While digitalis is being administered in these cases, the amount of urine excreted daily should be measured carefully. This is especially important when dropsy is present. Increase in the urinary excretion indicates that the action of the digitalis is satisfactory; but if the urine be scanty and no increase take place even on pushing the digitalis, the drug should be discontinued for a time, and tried again later, after further measures have been taken to relieve the venous engorgement.

When the dropsy is severe and persistent, the insertion of Southey's tubes in the lower portion of the legs may be of service to drain off the excess of lymph in the tissues.

In cases of slight mitral incompetence due to loss of tone of the cardiac muscle after such causes as acute illness, overwork, or exertion when out of condition, it is not usually advisable to insist on complete rest in bed; but graduated walking exercise for short periods of time gradually increased from day to day, with rest in the recumbent position for at least one hour before meals, may be ordered. At the same time, small doses of digitalis (5-10 min. of the tincture) with *nux vomica* or liq. strychninae may be given three times a day to improve the tonicity of the heart-muscle. A bracing dry climate is also of great importance in improving the general muscular tone. If anæmia be present, it should be treated with appropriate remedies. In cases of severe mitral incompetence resulting from pericarditis in which the pericardium has become adherent, rest in bed for a period of from two to three months or more is advisable, and a very quiet life should be led for at least a

year. In such cases the heart-muscle has, in all probability, been seriously damaged, and a long time must elapse before regeneration and efficient hypertrophy can take place. Every precaution must be taken to avoid the recurrence of cardiac rheumatism to which these young patients are liable; if possible, the winter should be spent in a warm, dry climate.

### MITRAL STENOSIS

**Etiology.**—Mitral stenosis, or constriction of the mitral orifice, is usually a sequela rather than an immediate result of acute endocarditis. The constriction of the mitral orifice is due to adhesion of the damaged valves and cicatricial contraction of the fibrous tissue formed in and around the valves and chordæ tendinæ during the process of repair. Some little time, therefore, must elapse before the condition is established; afterwards, in many cases, it appears to be slowly but persistently progressive.

Statistics show that it is more common in females than in males, an incidence of which no adequate explanation is forthcoming.

While the severe cases are the result of acute endocarditis, it is probable that some, of minor degree occurring in later life, are attributable to chronic degenerative changes and thickening of the mitral valves.

Though mitral stenosis is frequently met with in childhood and adolescence, the period at which the graver symptoms set in is usually rather later in life, between the ages of 20 and 30. The explanation of this would seem to be that the narrow rigid orifice cannot expand *puri passu* with the development of the heart, and the relative disproportion becomes a more serious handicap with advancing years.

**Morbid anatomy. The heart.**—The left ventricle is usually relatively small; the left auricle and the right ventricle are dilated and hypertrophied. The condition of the mitral orifice varies greatly. In extreme cases the orifice may be a narrow slit about  $\frac{1}{4}$  in. by  $\frac{1}{4}$  in., "button-hole" in character, with a rigid, fibrous, and, it may be, calcified margin, the valves as such having entirely disappeared. Frequently this slit is at the top of a funnel-shaped projection. In other cases, a circular rigid orifice admitting the finger may be present. Whatever the degree of stenosis, the valves are usually so rigid and adherent as to be incapable of functioning efficiently, if at all.

**Physiological and pathological effects of mitral stenosis.**—As the constriction at the

## VALVULAR DISEASE, CHRONIC

mitral orifice offers resistance to the flow of blood through it, the blood becomes dammed back in the left auricle, which becomes dilated. To overcome this resistance and to force the blood more rapidly through the narrowed orifice, the left auricle undergoes hypertrophy; but the degree to which hypertrophy can take place in this thin-walled structure is very limited, so that it soon becomes over-distended and the blood is dammed back in the pulmonary circulation. To meet this, the right ventricle hypertrophies and drives the blood through the lungs with greater force; thus the pressure in the pulmonary circulation is greatly increased, as also is that in the left auricle. It will be seen, therefore, that the main work of compensating for or counteracting the effects of the mitral lesion falls on the right ventricle, and the greater the constriction of the mitral orifice, the greater the demand for hypertrophy and increased driving power on the part of the right ventricle to hasten the flow

in which the circulation is brought to a standstill in the face of the prolonged and intense back-pressure, when right ventricular failure sets in. Thus, one or more wedge-shaped infarcts, often of very large size (2 or 3 in. across at their base), are frequently found post mortem.

**The liver.**—The so-called “nutmeg liver” is more frequently met with in mitral stenosis than in any other condition. The liver is greatly enlarged, engorged with blood, and on section the congested and distended intralobular veins stand out as small red areas surrounded by yellowish opaque rings of cells, atrophied and in a state of fatty degeneration. This combination gives rise to the so-called “nutmeg” appearance. The mechanism of its production is as follows: When the right ventricle becomes unequal to the task of maintaining the ever-increasing high pressure in the pulmonary circulation and becomes dilated, tricuspid incompetence sets in, with reflux of



Fig. 105.—Pulse of extreme mitral stenosis.

of blood through the narrowed orifice. For the right ventricle may be compared to a pump, the narrowed mitral orifice to a nozzle at the end of a hose pipe, the hose pipe being represented by the vessels and capillaries of the pulmonary circulation. It is obvious that the rise in pressure in the pulmonary circulation will give rise to great congestion, and entail serious damage to the parts of least resistance, namely, the capillaries, and in severe cases we find marked evidence of this post mortem.

The lungs in old-standing cases are tough, inelastic, firm on section, and of a peculiar brownish tint, a condition known as “brown induration.” Microscopically, the capillaries are seen to be engorged, tortuous, and bulging into the cavities of the alveoli, many of which are collapsed and obliterated, whilst others are filled with red blood-cells or their debris from ruptured capillaries. The lymphatics are loaded with pigment derived from altered hæmoglobin, which gives the peculiar brownish tint.

**Infarction.**—In the later stages of mitral stenosis infarction is very liable to occur from stasis and thrombosis in the smaller arteries,

blood into the right auricle, which becomes engorged and dilated. The blood in the inferior vena cava can with difficulty find its way into the already partly-filled right auricle, and this back-pressure becomes manifest in the hepatic veins. The liver thus becomes engorged and the intralobular veins and capillaries entering them in the centre of the lobules are distended with blood, whilst the intervening cells atrophy from pressure or undergo fatty degeneration.

**Physical signs. The pulse.**—The artery at the wrist is small and full between the beats; the pulse, small and of moderate tension, is usually regular in force and frequency till the later stages in which compensation is failing or auricular fibrillation sets in, when it becomes very irregular. The size of the artery and pulse wave is an important indication of the degree of stenosis, as the vascular system contracts down and adapts itself proportionately to the diminished output from the imperfectly filled ventricle (Fig. 105).

**The heart.**—The apex beat is usually displaced outwards to a varying degree by the hypertrophy of the right ventricle, but not

## VALVULAR DISEASE, CHRONIC

downwards to any extent, as the left ventricle is not usually enlarged. It is not forcible or thrusting, but resembles rather a short localized tap.

A presystolic thrill, over and just internal to the apex, can frequently be felt.

The right ventricle is hypertrophied. Epigastric pulsation is frequently present. The area of cardiac dullness is increased and may extend to the right of the sternum from dilatation of the right auricle. It is also increased outwards along the third and fourth left spaces, and upwards to the third or second intercostal space, by dilatation and hypertrophy of the left auricle, and of the conus arteriosus of the pulmonary artery.

The *most important sign of mitral stenosis* is a presystolic murmur audible just internally to the apex and usually vibratory or rumbling in character. It terminates abruptly in the first sound, which is short, sharp, and rather high-pitched. The second sound is almost invariably reduplicated at the apex, and the pulmonic second sound at the base is accentuated. This combination is very striking and characteristic, but is only present in the earlier stages of the disease. Later on, as compensation fails, the presystolic murmur may disappear because there is not sufficient power in the auricular systole to force the blood rapidly enough through the narrowed orifice to generate a murmur.

The short, sharp snapping first sound in which the presystolic murmur terminates, although very typical, is not easy to explain. The filling of the ventricle through the narrowed orifice takes longer than normal, and although diastole is prolonged, it is probable that the ventricle is imperfectly filled. At the commencement of systole, therefore, the ventricular walls, meeting little resistance, close down rapidly till they are suddenly brought up and made tense as they encounter the contained blood. This sudden tension of the muscular walls and the shortened systole might account for the short and sharp first sound and also for the sharp tapping apex beat.

The accentuation of the pulmonic second sound and the reduplication of the second sound at the apex are due to the high pressure in the pulmonary circulation which causes the pulmonic valves to close sharply and before the aortic valves.

The presystolic murmur may be short or may occupy the whole of the diastolic interval. More rarely it may be broken, and instead of

a continuous rumble there may be a short diastolic murmur in the early part of diastole as the ventricle actively expands and the blood is sucked in through the narrow orifice; then comes a short pause followed by a presystolic murmur leading up to the first sound, as the systole of the auricle takes place.

In a later stage the second sound may not be audible at the apex, but only the presystolic murmur and the first sound. The terminal stage is marked by extreme irregularity of the pulse and by the disappearance of the presystolic murmur. Then a confused medley of sounds may be heard at the apex, a short, sharp first sound, or a systolic murmur, or a first sound and systolic murmur, occurring at irregular intervals. It is not always easy under these conditions to diagnose the case as one of mitral stenosis from the physical signs, unless a presystolic murmur occurs from time to time, as sometimes happens, or the previous clinical history is known. This stage marks the breakdown of the right ventricle and paralysis of the left auricle, and the onset of auricular fibrillation. The presystolic murmur disappears because the auricle has given out and there is no longer an auricular systole to generate it.

The systolic murmur is indicative of mitral reflux; this occurs because the pressure maintained in the left auricle is no longer sufficient to resist regurgitation during systole, the right ventricle having failed.

The mitral ring being rigid, its "button-hole" orifice cannot be closed during systole; in the preterminal stages it is only the pressure maintained in the left auricle by the powerful right ventricle that prevents reflux. If the pressure in the left ventricle exceed that in the auricle, reflux takes place and a systolic murmur results; if the pressure in the auricle equal that in the ventricle, a first sound only is present. If, as occasionally occurs, the expiration effect of the ventricular expansion during diastole causes the blood to flow through the narrowed orifice sufficiently rapidly, a presystolic murmur may be heard from time to time in spite of the absence of the auricular systole. We have thus an explanation of the confused medley of sounds that may be met with.

The extreme irregularity of the heart-beat at this stage is attributed at the present day to what is known as auricular fibrillation, as demonstrated by electrocardiographic tracings in which the normal auricular wave is absent and is replaced by a series of rapid small

## VALVULAR DISEASE, CHRONIC

oscillations. The explanation given is that in this condition the dilated auricle ceases to beat co-ordinately, but that there is inco-ordinate contraction of its individual fibres. Thus, instead of a regular rhythmic stimulus being transmitted from auricle to ventricle, numerous feeble inco-ordinate stimuli are transmitted, to some of which the ventricle responds at irregular intervals, so that its rhythm is very irregular in force and frequency. The question of auricular fibrillation, however, is fully discussed under its own name, and does not enter into the scope of this article.

Fullness and distension of the veins in the neck is conspicuous, and, in the later stages when the tricuspid valve becomes incompetent, well-marked pulsation of the ventricular type is present.

The liver may be so greatly enlarged that its lower edge reaches to the umbilicus or well below it. At first the enlargement may be temporary, due to venous engorgement from a breakdown of compensation, when it may recover to an astonishing degree as the result of rest in bed and suitable treatment. The size of the liver is an important index of the amount of venous engorgement, and also of the condition of the right ventricle and the efficiency with which it is dealing with the strain thrown upon it by the constant and increasing high pressure in the pulmonary circulation. Careful examinations of the liver and palpation to ascertain its lower margin should never be omitted.

In the later stages the liver may be permanently enlarged from secondary fibrosis, though still fluctuating in size according to the degree of engorgement. Definite pulsation of the organ is often present when tricuspid incompetence has become pronounced.

**Symptomatology.**—Up to a fairly advanced stage few symptoms are present, and the patient is able to go about his or her ordinary avocation without any inconvenience save shortness of breath on undue exertion. As time goes on, the capillaries over the cheek-bones become congested and prominent, giving rise to a ruddy colour which might be taken for a sign of good health were it not contradicted by the dusky tinge of the lips. The cyanosis tends to become more marked and the shortness of breath more troublesome and pronounced as the aerating capacity of the lungs is diminished by the partial obliteration of alveoli due to the congestion caused by the constant high pressure in the pulmonary cir-

ulation, as explained in the section on morbid anatomy.

Hæmoptysis is fairly common, small pellets of dark blood being coughed up from rupture of congested capillaries. It is never profuse or alarming at this stage. In the terminal stages, however, when it is due to infarction of the lung, it may be very profuse and repeated.

Attacks of tachycardia setting in abruptly and lasting a few hours or some days, may be a serious and distressing feature and tend to recur. In two cases of moderate stenosis I have seen death result from these attacks. Irregularity or intermittency of pulse due to ventricular extrasystoles may also occur.

Cough is often a troublesome symptom, and from the congested condition of the lungs the patient is especially liable to attacks of bronchitis or bronchial catarrh.

Dropsy of the lower extremities does not usually set in till the terminal stage, but ascites is common, and often occurs at a comparatively early stage of the more serious symptoms as a result of the prolonged and severe engorgement of the liver.

**Diagnosis.**—In the first stage, when the presystolic murmur leading up to a short, sharp first sound followed by a reduplicated second sound is present, there is little difficulty in arriving at a diagnosis. In many instances the aspect of the patient, with injected capillaries over the cheek-bones and a cyanotic tinge of the lips, is so typical that, from a glance at the face, confirmed by finding a small thready artery at the wrist with a regular pulse, one can arrive at a diagnosis of mitral stenosis before proceeding to examine the heart.

A presystolic murmur, the so-called Austin Flint murmur, is met with in association with aortic incompetence, but the presence of the diastolic murmur at the base and a collapsing pulse will readily differentiate it. The differential diagnosis between this and mitral stenosis as a complication of aortic incompetence has been discussed under the latter (p. 493).

A short rumbling presystolic murmur is often met with during or shortly after an attack of cardiac rheumatism in children, but it is not of the typical vibratory character and does not terminate abruptly in the first sound, which instead of being short and sharp is rather muffled and frequently accompanied by a systolic murmur. Moreover, mitral stenosis does not set in till healing of the damaged valve

## VALVULAR DISEASE, CHRONIC

has taken place and cicatricial contraction has resulted.

In the terminal stage of so-called auricular fibrillation, in which there is extreme irregularity of pulse and the presystolic murmur has disappeared, as has already been explained, it may be a matter of considerable difficulty to arrive at a correct diagnosis in the absence of the earlier clinical history of the case. The result of efficient treatment by digitalis will, as a rule, soon clear this up, as a remarkable recovery usually takes place in favourable cases, and the presystolic murmur reappears as the rhythm becomes more regular.

**Prognosis.**—As regards long life, the prognosis is not very favourable, and from statistics of a number of cases at St. Mary's Hospital the average age of death was 33 for males and 38 for females.

One reason for the serious outlook in mitral stenosis is that if the lesion be established in childhood or early adolescence and the valves become cicatrized and adherent, the stenosed orifice cannot increase in size though the growth of the heart continues; thus the relative disproportion between the mitral orifice and the cavities of the heart increases as the heart attains its full development. Another reason is the progressive tendency of the lesion, for the contraction and rigidity of the mitral orifice tend to increase from cicatricial contraction; frequently calcification of the valve takes place from deposit of lime salts in the damaged tissues. The damage to the lungs by the high pressure in the pulmonary circulation with the consequent diminution of their aerating capacity is also a serious factor. As regards prognosis, the size of the radial artery at the wrist, the degree of enlargement of the right ventricle, the aspect of the patient, and the readiness with which cardiac symptoms are induced on exertion, afford useful information as to the degree of severity of the lesion.

If the pulse be small and wiry, the right ventricle enlarged and hypertrophied so that epigastric pulsation is present, the face injected and the lips cyanosed, and dyspnoea set in on slight exertion, one should infer that the degree of contraction is considerable and the outlook consequently unfavourable.

It is advisable to examine the liver in all cases, as enlargement of this organ from venous engorgement indicates that the right ventricle is beginning to fail.

In the later stages, when serious symptoms, such as cyanosis, orthopnoea, ascites, pulsation

of the liver and of the veins in the neck, and irregularity of the pulse are present, a temporary recovery need not be despaired of with rest in bed and suitable treatment, if imprudence, over-exertion, or an attack of bronchitis be responsible for their onset. If, however, a similar breakdown has occurred previously and these serious symptoms set in while the patient is at rest in bed, the prognosis is necessarily very unfavourable.

**Treatment.**—In the early stages no special treatment is required; but in view of the tendency to progressive cicatricial contraction of the damaged valve, every precaution should be taken against conditions which tend to irritate or injure further the affected valve. Such are anæmia, overstrain, rheumatism, or auto-intoxication which may result from constipation or a diet rich in purin bodies. Bronchitis, also, should specially be guarded against, because it puts additional strain on the already overtaxed right ventricle.

When there is marked evidence of venous stasis in the lungs such as cyanosis and dyspnoea, with engorgement or pulsation of the veins in the neck and enlargement of the liver, indicating that the right ventricle is failing and unable to maintain the pulmonary circulation efficiently, rest in bed is, of course, imperative. If the condition has been brought about by an attack of bronchitis, venesection will be of striking service. In all cases a mercurial purge, calomel or pil. hydrargyri with colocynth and hyoscyamus, should be given, followed up by a saline in the morning. Digitalis may then be administered with marked benefit in doses of 15 min. of the tincture every four hours for two or three days, and afterwards three times a day, but should not be persisted with too long. The amount of urine excreted should carefully be noted.

In the final stages, in which complete arrhythmia or auricular fibrillation is present, digitalis in rather larger doses is of great service. The effect of digitalis is to slow the heart, and, in this condition, when administered in sufficient doses, it also produces a certain degree of heart-block, a large proportion of the impulses transmitted from the fibrillating auricle being arrested. The pulse being thus slowed and diastole lengthened, a longer time is allowed for the ventricle to fill. Such is the present-day explanation, but it must be recollected that digitalis also increases the tonicity of the heart-muscle. In mitral stenosis it will have a greater effect on the

## VALVULAR DISEASE, CHRONIC

hypertrophied right ventricle than on the small left ventricle, and will be all-important in enabling the right ventricle to regain its tonicity and deal more effectively with the congested pulmonary circulation. This I consider of great importance. The drug may be given in large doses (15–20 min. of the tincture, or  $\frac{1}{16}$  gr. of Nativelle's digitalin, every four hours), but should not be pushed too far or too long. Three days is usually sufficient to obtain the cumulative effect. Indications of overdosing are extreme slowing of the pulse and the appearance of "coupled beats" in groups of a normal beat followed by an extrasystole.

Remarkable temporary recoveries may take place from this condition even after one or two breakdowns; but the response to treatment is not so satisfactory as in mitral incompetence, for the narrow mitral orifice constitutes a fixed obstacle which maintains an unremitting resistance to the outflow from the pulmonary circulation, and makes it difficult for the right ventricle to overcome the ever-increasing strain to which it is subjected.

When infarction of the lung occurs, the end is near, and treatment is of little avail.

### MITRAL STENOSIS WITH INCOMPETENCE

Frequently we meet with mitral stenosis combined with incompetence—that is to say, we hear at the apex a presystolic murmur leading up to a short, sharp first sound followed by a systolic murmur. The presystolic murmur is usually rather short and not of the prolonged rumbling type, and the first sound is not replaced to any extent by the systolic murmur.

In these cases the mitral orifice is never of the extreme "button-hole" character, but the stenosis is slight or moderate in amount and regurgitation predominates. The presystolic murmur may arise from slight obstruction caused by the thickened scarred edge of a mitral flap, which has been damaged by endocarditis, projecting into the blood-stream during diastole. In more pronounced cases it may be due to adhesions between the cicatrized mitral valves, which, while causing some obstruction to the inflow from the auricle during diastole, leave the mitral orifice widely patent during systole.

The prognosis in cases of this class is more favourable than in pure mitral stenosis, and is rather that of mitral incompetence due to endocarditis.

### AFFECTIONS OF THE PULMONARY VALVES

Acquired lesions of the pulmonary valves are extremely rare, as rheumatic endocarditis of the right side of the heart is not common, and if it does occur the tricuspid valve is much more prone to be attacked than the pulmonary. **Pulmonary stenosis** is almost invariably the result of a congenital heart lesion, and is dealt with elsewhere (see HEART, CONGENITAL DISEASE OF). **Pulmonary incompetence** is very rare, but Barié has collected records of 50 cases with autopsies. In 40 per cent. of his cases it was of congenital origin, in 16 per cent. it was the result of rheumatic endocarditis. When present it gives rise to a diastolic murmur over the second intercostal space to the left of the sternum, but, as this is met with in aortic incompetence, the differential diagnosis must rest on the absence of any of the physical signs commonly met with in aortic incompetence, viz. collapsing pulse, pulsation of carotids and capillaries, and hypertrophy of the left ventricle. There may be visible pulsation of the conus arteriosus in the second left intercostal space. If the lesion is severe there will be dilatation and hypertrophy of the right ventricle, with cyanosis and dyspnoea on slight exertion.

**Functional murmurs.**—A soft blowing systolic murmur over the pulmonary area is frequently met with in anæmia, and in cases of cardiac dilatation associated with loss of tone of the cardiac muscle. The pulmonary artery also shares in this loss of tonicity, so that slight dilatation beyond the valves results. The murmur may thus be explained as due to a slight virtual obstruction as the blood flows through the valves into the somewhat wider channel beyond. The murmur may last some weeks, or even months, but disappears as the heart recovers its tonicity. This I have observed in a number of cases.

### AFFECTIONS OF THE TRICUSPID VALVE

**Tricuspid incompetence** due to dilatation of the right ventricle, secondary to mitral lesions and increased pressure in the pulmonary circulation, is of very common occurrence. Apart from mitral lesions, it may result from obstruction to the pulmonary circulation by emphysematous and fibroid changes in the lungs associated with asthma and chronic bronchitis. This is more common in later life when there is commencing degenerative change in the heart-muscle. It is also frequently met with in relapsing rheumatic pericarditis, in

## VALVULAR DISEASE, CHRONIC

which the dilatation of the right ventricle may be very great, especially when the pericardium becomes adherent to the damaged heart-muscle.

Other and rarer causes of tricuspid incompetence are pulmonary stenosis, or rupture of an aortic aneurysm into the pulmonary artery.

Organic lesions of the tricuspid valve resulting from endocarditis are rare. When present they are more commonly the effect of malignant than of rheumatic endocarditis. In cases which survive, presumably of rheumatic origin, tricuspid incompetence seldom, if ever, occurs as an isolated lesion, but is associated with disease of the mitral or aortic valve.

**Physical signs and symptoms.**—The effect of tricuspid incompetence is to cause engorgement of the right auricle and obstruction to the venous return. The veins in the neck become distended with blood, and pulsation of the ventricular type may occur; the face is congested. From obstruction to the return of blood by the inferior vena cava the liver becomes engorged and enlarged, and its lower edge may extend down as far as the umbilicus or even below. Frequently in severe cases pulsation of the liver is present.

In the later stages, œdema of the lower extremities occurs and ascites may be present. The patient is cyanosed and short of breath. The jugular pulse is altered in character; the auricular wave disappears in advanced cases, and the tracing assumes the ventricular type, as there is free communication between the auricle and the ventricle during systole.

The right ventricle of the heart is usually enlarged and dilated when the incompetence is secondary to mitral disease and epigastric pulsation is present. A systolic murmur is usually audible over the lower end of the sternum and to the left of it, but it is by no means always present, and when present is not always distinguishable from a conducted murmur. It is, however, of little importance for diagnostic purposes, since the enlarged pulsating liver and jugulars are pathognomonic of the condition.

**Treatment.**—Complete rest in bed with free purgation and, if necessary, venesection or the application of leeches over the liver, followed by the administration of digitalis, is indicated. It is in cases of right-ventricle failure secondary to mitral disease that digitalis is especially called for, and the results obtained are very striking and most successful, unless the muscular wall of the right ventricle is degenerated or too much damaged to respond to treatment.

## VARICOCELE

### TRICUSPID STENOSIS

This is a comparatively rare lesion, and when present is usually associated with other valvular lesions, most commonly with mitral stenosis, but sometimes with mitral and aortic stenosis.

The changes in the tricuspid valve are similar to those which take place in the mitral valve in mitral stenosis. As a result of the obstruction the blood is dammed back in the right auricle, which dilates and then hypertrophies. The auricular wave in the jugular pulse is more pronounced at first, but eventually the right auricle becomes paralysed and the wave disappears. Since there is great obstruction to the venous return, the jugular veins become distended, the liver engorged, and œdema is produced. The patient is cyanosed and short of breath from an early period. The auscultatory sign of tricuspid stenosis is a presystolic murmur over the base of the sternum, but, as the right auricle soon gives out, and when this takes place there is no compensatory mechanism to maintain a pressure sufficient to generate a presystolic murmur, it is often absent, so that the condition is very seldom diagnosed during life. It is, moreover, nearly always associated with other valvular lesions, most commonly with mitral stenosis, which adds to the difficulty of diagnosis. Of 114 cases collected by Leudet, diagnosis before death was made in only 6.

**Prognosis.**—This is eminently unfavourable, as no compensatory changes can take place to deal with the venous stasis in the right auricle, which is reflected back into the great veins.

**Treatment** must be mainly palliative, as little can be done to relieve the stasis in the left auricle, except rest in bed, free purgation, and venesection from time to time.

JOHN F. H. BROADBENT.

**VAQUEZ'S DISEASE** (see CYANOSIS.)

**VARICELLA** (see CHICKENPOX).

**VARICOCELE.**—A pathological dilatation of the pampiniform plexus of veins. This plexus is situated in the spermatic cord in the male and in the broad ligament in the female, and issues into the spermatic or ovarian vein according to the sex.

Varicocele is met with chiefly in males, and is usually only found on the left side of the body.

**Etiology.**—The angle at which the left spermatic vein enters the left renal vein, and

## VARICOSE VEINS

the pressure exerted by the iliac colon on the spermatic vein, are two possible factors in the causation of the condition. Abdominal tumours or malignant glands pressing on the renal of the spermatic vein may cause a varicocele on either side.

**Pathology.**—The dilated veins of the spermatic cord increase in length and become tortuous, and the left side of the scrotum becomes lax.

**Symptomatology.**—An aching pain is usually complained of, but many cases have no symptoms. Examination reveals a swelling of the left side of the scrotum extending up to the external abdominal ring, disappearing when the patient lies down, and on palpation giving a sensation to the fingers as if the scrotum were "a bag of worms." In the female, dilatation of the pampiniform plexus causes an aching sensation, relieved on lying down, in a patient who has no signs of prolapse.

**Diagnosis.**—Consideration of the above symptoms makes diagnosis easy. In the male a right-sided varicocele, or one developing suddenly, should cause one to look for tumour or glands pressing on the right spermatic vein. It is possible that some cases of right-sided dilatation of the veins in the broad ligament are mistaken for appendicitis.

**Treatment.**—Since no serious inconvenience is caused by a mild degree of varicocele, either the condition may be ignored or a suspender may be worn. If aching pain is troublesome, or if the patient is going to the tropics, or is desirous of entering any of the public services, he may wish for operation.

Operative treatment is simple, and consists in making a small incision over the external abdominal ring, incising the cremasteric fascia, carefully separating the veins of the spermatic cord from the vas deferens, ligating the veins in two places, cutting across the veins between the ligatures, and suturing the two severed ends so as to shorten the cord and pull up the testis. Some Continental operators also excise a portion of the scrotal skin.

ZACHARY COPE.

**VARICOSE ANEURYSM** (*see* ANEURYSM, ARTERIO-VEINUS).

**VARICOSE VEINS.**—A varicose vein, or varix, is an abnormally dilated venous channel. In this place only dilated superficial veins are considered, dilated veins of the spermatic cord being dealt with in VARICOCELE, and dilated hæmorrhoidal veins in HÆMORRHOIDS.

**Etiology.**—There is a considerable variation within normal limits in the size of the superficial veins. In some they are congenitally enlarged. The main causes of varicose veins are various hindrances to the free return of the blood to the heart. These hindrances may affect the main venous trunks in the abdomen or thorax, the deep veins of the limbs, or the superficial veins themselves. In those who have to stand for long periods, gravity may dilate the veins of the lower extremities.

Pressure upon the main veins of the abdomen by tumours, enlarged glands, or fæcal accumulation may cause the distal veins to become distended. The enlarging pregnant uterus frequently causes acute varicosity of the veins of the legs and vulva, especially in young primiparæ. The wearing of tight garters tends to produce or increase a varicosity of veins of the leg.

If the deep veins of the trunk or limbs are thrombosed or are occluded for a long time (as by the sustained muscular effort of athletes), compensatory enlargement of the superficial veins occurs. Thus, in thrombosis of the inferior vena cava a chain of large veins communicates over the abdomen and thorax from the femoral veins to the superior epigastric and other tributary veins of the superior vena cava.

**Pathology.**—The pathological condition is best studied in the veins of the leg, where either the internal or the external saphenous vein may be affected. The vein is much dilated, frequently tortuous and greatly increased in length, and the valves are incompetent. At certain intervals bulbous dilatations may be noticed. There is thickening of the tissues around the vein owing to the periphlebitis. Firm clots can sometimes be felt in the vein; and phleboliths may be produced by their calcification.

### Symptomatology and complications.

In mild cases the only symptom complained of is an aching of the legs and feet after standing or walking. A slight degree of swelling and oedema of the lower part of the leg is often seen. In old-established cases one or more of the following complications may ensue, viz. hæmorrhage, eczema, ulceration, thrombosis, phlebitis. *Hæmorrhage* generally occurs from a vein which has by constant pressure forced its way through the thinned skin and sustained some slight traumatism. Fatal hæmorrhage has many times occurred, although direct pressure on the bleeding-spot is sufficient to stop it. Varicose *eczema* is usually found in



## VARICOSE VEINS

the lower half of the leg. Hæmorrhage seldom occurs from eczematous patches.

The **diagnosis** of varicose superficial veins is easily made by inspection when the patient stands upright. Occasionally, some difficulty may be found in distinguishing between a femoral hernia and a varix of the upper end of the internal saphenous vein, which may form a rounded swelling just over the femoral canal. Such a varix gives an impulse and a thrill on coughing, disappears when the patient lies down, and must be suspected when varicose veins are observed lower down the limb.

**Treatment.**—In the case of varicose veins of the lower extremities, all impediments to free venous return must be removed. Elastic or other tight garters must not be worn. Constipation must be avoided, and abdominal examination must be made in order to detect any tumour which might press on the venous trunks. Prolonged muscular exercise tending to force the blood from the deep to the superficial veins (e.g. cycling) should be avoided. An elastic or semi-elastic support should be worn. If an elastic stocking is used it should be well ventilated to allow evaporation of perspiration. A bandage of doanett or crêpe (velpeau) wound firmly round the limb from the foot upwards serves well to support the veins. Massage is useful to prevent œdema.

Operation for varicose veins in an otherwise healthy young adult should be advised—

(1) When pain and discomfort prevent the patient undertaking ordinary active exercise.

(2) When the veins form thin-walled dilata-tions adherent to the skin and threatening hæmorrhage; *a fortiori* if hæmorrhage has occurred.

(3) When eczema or ulceration threatens.

(4) When there is a localized patch of throm-bosed superficial veins.

(5) When the patient wishes to enter the public services. In general, if the varicose veins prevent the patient from earning his living, operation is to be advised.

In middle-aged and elderly people operation is much more rarely undertaken.

It consists in excising the parts of the vein which are varicose. Either a few long incisions are made, allowing long portions to be excised, or many small incisions, permitting small segments to be removed. It is some-times necessary to remove adherent overlying skin.

## VEGETATIVE SYSTEM

In addition, in bad cases it is advisable to expose and divide between two ligatures the internal saphenous vein at the saphenous opening.

ZACHARY COPE.

**VARIOLA** (*see* SMALLPOX).

**VEGETABLE POISONS** (*see* POISONS AND POISONING).

### VEGETATIVE (SYMPATHETIC AND PARASYMPATHETIC) SYSTEM, DISEASES OF.

— The vegetative system, in contradistinction to the voluntary or somatic nervous system, innervates the unstriated muscles of the viscera, the muscles of the vascular system including the heart, the secretory tissues, and certain striated fibres of the heart and œsophagus; it also innervates the genito-urinary apparatus. It is therefore the nervous system controlling all such structures as are capable of automatically continuing their functions independently of the central nervous system. It is convenient to consider this automatic nervous mechanism as a whole under the designation of the vegetative nervous system. Comprising two systems presenting some anatomical and physiological differences—the *sympathetic* and the *para-sympathetic* systems—it is characterized by certain structural differences from the plan of the somatic nervous system. Whilst the efferent fibres that innervate the voluntary muscles arise from nerve-cells in the cerebro-spinal axis, the efferent fibres of the vegetative system arise from nerve-cells that lie without the central nervous system, either in definite chains of ganglia, or in terminal ganglia situated still more peripherally on the course of the nerves as they enter the viscera. These efferent neurones are connected with certain afferent fibres of the posterior roots by communicating neurones that spring from the cells in the lateral horn of the cord, and pass through the anterior roots as a white ramus communicans, to end in synapses round nerve-cells either in the great ganglia of the sympathetic chain or in the terminal ganglia. The arrangement is not so clear in the parasympathetic system, but it obtains nevertheless. Its cells lie in terminal ganglia; that is, they have their station close to the structures they innervate. The connecting fibres of its cranial portion run in the 3rd nerve to the cells of the ciliary ganglion which controls constriction of the pupil; in the 7th nerve and through the chorda tympani to Langley's and the sublingual gan-

## VEGETATIVE SYSTEM, DISEASES OF

glia which supply the submaxillary and sublingual glands; and in the 9th nerve to the otic ganglion which innervates the parotid gland. But the main portion of the bulbar parasympathetic runs in the vagus to the ganglia innervating the heart, lungs, liver, pancreas, stomach, and intestines. The sacral parasympathetic neurones run in the pelvic nerve to the ganglia that innervate the descending colon, rectum, anus, bladder, and genital organs.

The accompanying diagram (Plate 41), after Meyer and Gottlieb, shows the distribution of the sympathetic and parasympathetic systems. It will be at once seen that practically all autonomic structures, with the exception of the sweat-glands, receive a double innervation—from both sympathetic and parasympathetic systems. These two divisions of the vegetative nervous system react differently to chemical stimulation. Adrenalin acts on all the sympathetic nerve-endings, causing vaso-constriction; it augments the action of the excitator nerves of the heart, causing a strengthening and acceleration of the pulse, dilates the pupil, and enhances the secretory activity of the glands. Where the sympathetic nerve-endings have an inhibitory action on unstriated muscle, as in the stomach, intestines, and bladder, adrenalin inhibits the automatic activity. The sudorific nerve-endings alone form an exception, as adrenalin has no effect on them. The parasympathetic nerve-endings are not affected by adrenalin, but they react to two groups of drugs which have no action on the sympathetic. One group, of which we may take pilocarpine as a type, stimulates the parasympathetic nerve-endings. By its action all glandular activity under control of the parasympathetic is augmented, and the pupil and the unstriated muscles of the bronchioles, the whole of the alimentary tract, the bladder, and the uterus contract. The sudorific nerves which are of sympathetic origin are, however, also stimulated, so that the antithesis between sympathetic and parasympathetic systems in relation to their behaviour to these chemical stimuli is not complete. Atropine, as the type of the second group of drugs, inhibits all forms of parasympathetic activity; secretion and the contractions of the unstriated musculature are inhibited, and the inhibitory action of the vagus on the heart is lost.

It will be noted that the activities of the sympathetic and parasympathetic systems have

a mutually antagonistic action—the inhibitory fibres of the one are neutralized by the excitatory fibres of the other, and vice versa. This consideration is of the greatest importance in evaluating the effects of lesions of the sympathetic and parasympathetic, inasmuch as loss of function of the one system will not be followed by purely deficiency symptoms but by over-action of the other.

**Local lesions.**—Uncomplicated local lesions of the vegetative system are rare, and the symptomatology of thoracic and abdominal disturbances due to them is, as a rule, obscured by other factors.

Paralysis of the *cervical sympathetic* occasionally occurs either as a result of injury or from pressure tumours or aneurysms. The symptoms of the lesion are: Pseudoptosis or drooping of the eyelid; absence of dilatation of the pupil on shading the eye, though, owing to the parasympathetic fibres of the 3rd nerve being unaffected, the pupil contracts to light and on accommodation, and is, as a rule, smaller than that on the sound side; enophthalmos, due to paralysis of the muscle of Müller, makes the palpebral fissure look smaller; there is diminution of intraocular pressure; the homolateral side of the face is pale and, if pilocarpine be injected, does not sweat; the pupil does not dilate to a painful stimulus applied to the neck, and if the lesion be above the lower cervical ganglion it does not dilate to cocaine; if, however, the lesion be below the ganglion, the pupillary fibres escape and dilate normally. Irritative lesions of the cervical sympathetic give the reverse of this picture, being distinguished by exophthalmos, mydriasis, flushing and sweating, and retraction of the upper lid.

The *angio-neuroses*, such as Raynaud's disease, erythromelalgia, angio-neurotic oedema, localized anidrosis or hyperidrosis, may be ascribed to local vegetative nerve-lesions, but their pathology is obscure.

**Pathology.**—The pathology of the vegetative nervous system is closely allied to that of the ductless glands. The chromaffin cells of the sympathetic paraganglia secrete adrenalin, which excites all sympathetic nerve-endings, while the thyroid secretion lowers the threshold of their excitability. The lymphatic tissue appears to secrete a hormone raising the excitability of the parasympathetic fibres. The internal secretion of the pancreas diminishes the excitability of the sympathetic system, while that of the pituitary raises it. There is

## VEGETATIVE SYSTEM

less definite evidence that the internal secretions of other glands have a local or general action on the vegetative nervous system.

Two general states of hyperexcitability of the vegetative nervous system are recognized—hyperexcitability of the sympathetic system or sympatheto-tonus, and hyperexcitability of the parasympathetic system or vagotonus. The use of these terms has been much abused, and how far these states of hyperexcitability can be considered as existing as permanent physiological conditions may be doubted, but it is true that under certain conditions, as yet not well understood, a patient may show signs of hyperexcitability of one or other. When the *parasympathetic* system is hyperexcitable we may have some or all of the following symptoms, viz. miosis, spasm of accommodation (excitation of the sphincter pupillæ and ciliary muscles), widening of the palpebral fissure, salivation and lachrymation (chorda tympani and lachrymal nerves), pallor (vaso-motor spasm), bradycardia and cardiac spasm (cardiac vagus), bronchial asthma (pulmonary vagus), increased peristalsis of the intestines, spasmodic contractions of the intestines and spastic constipation, tenesmus and stammering bladder (visceral branches of the vagus). There may also be eosinophilia; this blood-state is produced experimentally by drugs exciting the vagus. A patient who shows some or all of these signs of parasympathetic over-activity is generally found to be hypersusceptible to pilocarpine, and this idiosyncrasy has been used for diagnostic purposes. The frequent coexistence of signs of vagotonus with the lymphatic hyperplasia of status lymphaticus is suggestive in view of the alleged exciting influence of lymphatic extracts on the vagus. The condition of hyperexcitability of the *sympathetic* system is characterized by mydriasis, enophthalmos, tachycardia, and vaso-motor disturbances, as flushing, heat sensations, palpitation, glycosuria and visceroptosis. Following emotional disturbances, some signs of sympathetic hyperexcitability frequently occur combined with enlargement of the thyroid, though in such cases the occurrence of exophthalmos shows that the syndrome cannot be considered a purely sympathetic one. The excessive stimulation of the sympathetic system by emotional disturbance may lead to the over-action of the sympathetic mechanism responsible for the organic response to emotion, and this in a vicious circle to psychical dis-

## VELD SORE

turbances; hence psychotherapeutic measures in the treatment of neuroses whilst the bodily condition continues unchanged are doomed to failure.

Symptoms of over-action of the parasympathetic system may arise from impairment of sympathetic antagonism, as in Addison's disease, whilst in the early stages of tuberculosis there are many symptoms referable to its over-excitation, possibly as a result of lymphatic hyperplasia. It is possible to regard the secretory and vaso-motor constitutional disturbances at the climacteric as due to disturbances of the vegetative system caused by upset of the balance of the internal secretions. Increased excitability of the sympathetic system in some forms of diabetes and thyroidism may be demonstrated by the fact that instillation of adrenalin into the conjunctival sac stimulates the sympathetic nerve-endings and causes mydriasis, a reaction which is absent in the normal eye.

A separate pathology of the vegetative nervous system cannot, however, be formulated in the present state of our knowledge, nor can its specific disturbances be considered apart from the pathology of the internal secretions. But it is obvious that the vegetative nervous system constitutes the mechanism by which all symptoms of visceral origin are manifested in every disease. Thus, the visceral crises of tabes can be best explained by the selective action of the syphilitic toxin on the vegetative nervous system, and most trophic and motor disturbances of the automatic viscera occurring at points remote from an actual structural lesion can often only be accounted for by its mediation. Its pathology is thus coextensive with our knowledge of general pathology, and cannot be usefully considered separately.

F. L. GOLLA.

**VEINS, INFLAMMATION OF** (*see* PHLEBITIS).

**VEINS, VARICOSE** (*see* VARICOSE VEINS).

**VELD SORE** (*Syn.* Ulcerative dermatitis, Natal Sore).—An ulcerous condition of the skin seen in South Africa. In Australia a similar affection occurs, and is called "barkoo rot." The lesion has probably no specific organism as the causative agent, but is apparently a streptococcal manifestation. Streptococci are almost invariably found when searched for. The first sign is an itching papule. Shortly this becomes a vesicle and then a pus-

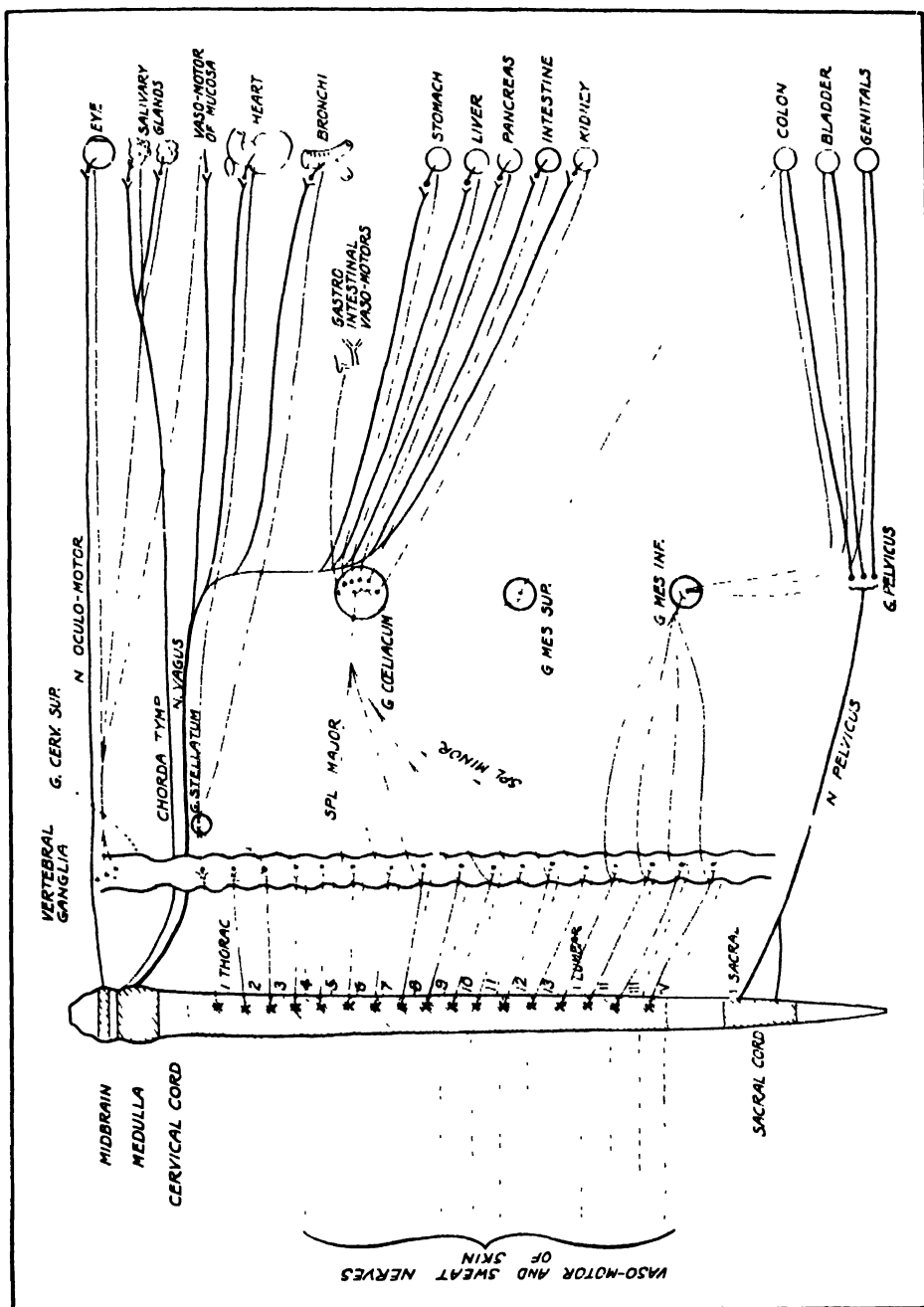


PLATE 41.—DIAGRAM OF THE VEGETATIVE NERVOUS SYSTEM. (After Meyer and Gottlieb,  
 "Pharmacologie," published by Urban & Schwarzenberg.)

Sympathetic system, red; parasympathetic system, blue.



## VENESECTION

tule, which ruptures, leaving a shallow ulcer that spreads somewhat and is covered in by a yellow crust. The condition is almost painless; it is itching that is chiefly complained of. The lesions usually appear upon the legs and feet, sometimes upon the hands or arms. The disease is rather resistant to treatment, and lasts from one to three months or even longer. In the absence of any indication of a specific causal organism, the ordinary treatment of a septic ulcer is to be employed. The removal of crusts by fomentations or soaking with sterile oil is followed by the washing of the wound with an antiseptic lotion and the application of some ointment such as ung. hydrarg. ammoniatum.

C. A. PANNETT.

**VENEPUNCTURE** (see BACTERIOLOGY AND PATHOLOGY, CLINICAL).

**VENEREAL DISEASES** (see GONORRHOEA; SYPHILIS; ULCUS MOLLE).

**VENESECTION.** (*syn.* Phlebotomy).—Venesection has fallen into unmerited desuetude on account of the reaction which followed its abuse in the eighteenth and early part of the nineteenth century. There are certain conditions in which it may be employed with benefit to the patient.

**Indications.**—1. *Engorgement of the systemic veins* in right-heart failure, whether due to valvular disease or consequent upon rapidly produced impediment to the pulmonary circulation. It is, thus, a useful procedure in the early stages of pneumonia, when there are evidences of dilatation of the right side of the heart. It is unsuitable for the weakly or the elderly, and is particularly indicated for the plethoric adult. Removal of as little as 5 oz. of blood will often relieve the embarrassed circulation of systemic engorgement sufficiently to give the patient sleep.

2. *Toxæmic states*, notably in uræmia, eclampsia, cholæmia, and diabetic coma, the object being to remove a proportion of the circulating poisons. In this regard it is often combined with saline infusion into a vein of the opposite arm in order to dilute the toxins which remain.

3. *High blood-pressure.*—In cases of arteriosclerosis and Bright's disease, when the blood tension is unduly high and is producing symptoms, venesection affords a means of quickly reducing it and relieving the symptoms. The effect, however, is often transitory, and other

measures should be employed subsequently. Venesection, by rapidly reducing blood-pressure, is a safeguard against cerebral hæmorrhage. When hæmorrhage has actually occurred it may be employed if the tension remains high, either in the early stages or if hæmorrhage continues, but it must be remembered that an increasing tension in cerebral hæmorrhage may represent an endeavour on the part of the vaso-motor centres to counteract increased intracranial pressure.

4. *Thermic fever.*—Venesection is particularly applicable to that variety of fever in which there is much engorgement and asphyxia.

5. In *hæmoptysis* due to traumatic lesions of the lung associated with severe hæmorrhage, as, for instance, damage to the lung by a fractured rib.

**Method.**—The vein selected is usually either the median basilic or median cephalic, though, in the case of children, or very fat people, choice is sometimes made of the external jugular. More rarely the internal saphenous is opened at the ankle. When a vein at the bend of the elbow is chosen, the patient should be sitting up with the arm hanging down, so that the veins are rendered prominent by swelling. A strip of bandage is then bound round the upper arm over a pad placed on the brachial vein, and tied by a knot over the pad. It should be bound firmly but not so tightly as to stop the flow of blood in the brachial artery. The arm is then raised to the horizontal position, and the patient is directed to grip a tight roll of bandage or some firm object of similar diameter. The muscular contraction increases the turgescence of the veins. The operator now steadies the vein and the skin over it by placing his thumb on the vein just below the site chosen for the incision, and with a sharp scalpel or lancet incises it obliquely. On removing his thumb, blood should spurt freely from the incision until some 5–8 oz. are removed. If more is required it can be obtained by firmly stroking the arm from below and upwards, and by directing the patient alternately to grip the bandage and release it. When the desired amount of blood has been removed the ligature must be untied, the arm raised, and a pad placed over the wound, where it is kept firmly in position by a figure-of-eight bandage. With proper care no complication should result; the two occasional accidents, sepsis and wounding the brachial artery, are easily avoided. The vein should not be opened blindly, and, if not plainly

## VERSION

visible, should be exposed very carefully by dissection.

Should the jugular vein be selected, the chief points to be observed are that the pad must be applied very firmly over the clavicle, external to the sterno-mastoid, and that the vessel should be incised in its long axis as it lies on the sterno-mastoid. The wound should be closed by another pad before that placed over the clavicle is removed. These precautions obviate the risk of air entering the vein.

FREDERICK LANGMEAD.

**VERONAL POISONING** (*see* POISONS AND POISONING).

**VERRUCA NECROGENICA** (*see* SKIN, TUBERCULOSIS OF).

**VERRUCÆ** (*see* WARTS).

**VERSION.** — Version is the operation of changing the lie of the child to produce a breech or vertex presentation—podalic and cephalic version respectively.

The three methods of performing version are :

1. *External version*, by abdominal manipulation only, before or very early in labour.

2. *Bipolar version*, a bimanual method, possible when the os will admit two fingers.

3. *Internal version*, performed when the whole hand can be introduced within the uterus.

1. **External version** is nearly always cephalic. The **indications** are—

(i) Malpresentation of the child, chiefly breech presentations in a primigravida, and occasionally in a multipara. In primigravida during pregnancy it is of particular importance to bring about a vertex presentation if the child is presenting by the breech, because, firstly, the fetal mortality in breech labour in primigravida is about 1 in 9 in all cases. Secondly, in cases of contracted pelvis it is not possible accurately to determine the proper date for induction of labour unless the degree of engagement of the presenting head can be tested by pushing it into the brim. Even in cases where the pelvic measurements are apparently normal, it is well to have the head presenting in all primigravida.

(ii) Shoulder presentations.

**Method.**—External version is only possible if there is sufficient liquor amnii to render the child easily movable, yet not so much as to make its manipulation difficult. Further, the abdominal and uterine walls must be lax.

Anæsthesia should be employed after failure without its help.

Difficult cases are those where the back lies posteriorly, and the head far back under the ribs, where the legs are extended, and also where the breech has descended low into the pelvis.

Failure to turn the child is common, but spontaneous version often follows the attempt.

The operation is carried out thus: After passing water, the patient lies in the Trendelenburg position. The operator stands at her side, on the same side as the child's back, and places both hands on the farther side of the breech, which is then drawn out of the pelvis towards him into the iliac fossa. The breech is held in this position by one hand while the other is applied to the head in the attempt to push it in the opposite direction by short, sharp jerks. While the head is thus driven down, the breech is pushed up until it has passed the uterine equator. After this point is reached it is easy by further manipulation of the head alone to bring it into the pelvic brim. When the operation is complete it is advisable to apply a tight binder for a few days.

2. **Bipolar version** is usually podalic. The **indications** are—

(i) Shoulder presentations.

(ii) Ante-partum hæmorrhage, especially placenta prævia, where it is not possible to use a de Ribes bag.

(iii) Face presentation which will not engage, or is complicated by a prolapsed cord or arm in which reposition has failed.

(iv) Flat pelvis, certain cases where the true conjugate measures more than  $3\frac{1}{2}$  in., especially when complicated by a face, brow, or cord presentation.

(v) Brow presentations.

(vi) Occasionally when rapid delivery is necessary, as in eclampsia.

**Method.**—The cervix should admit two fingers, and there should be just enough liquor amnii to render the child easily mobile, but not so much that the child slips up from the thrust of the fingers.

The patient is anæsthetized and catheterized, and preferably lying on the side opposite to that occupied by the back of the child. The gloved fingers are then passed through the cervix and by them the head is pushed away from the os so that the *abdominal* surface comes to lie over the operator's fingers, while the other hand presses down the breech in the

## VERTIGO

opposite direction. When the head has been pushed away the shoulder and elbow are similarly thrust after it, until the knee comes within reach of the fingers, which are then hooked over the popliteal space. In this way the leg is brought well down through the os, while the external hand pushes up the head.

**3. Internal version** is always podalic, and only possible when the os will admit the hand.  
**Indications—**

- (i) Shoulder presentations.
- (ii) Face and brow presentations, as above.
- (iii) Flat pelvis, as above.
- (iv) Placenta prævia, after expulsion of de Ribes' bag.
- (v) Prolapse of the cord, if the child is alive, and after expulsion of de Ribes' bag.
- (vi) Where rapid delivery is required, forceps being contraindicated.

The **contraindications** are important; they are—

- (i) Tonic uterine contraction, or when the uterine relaxation between the pains is imperfect.
- (ii) After the liquor amnii has been drained away, and the uterus has grasped the child. In these conditions the uterus will almost certainly be ruptured if version is attempted.
- (iii) Generally-contracted pelvis.
- (iv) Flat pelvis, where the true conjugate is less than  $3\frac{1}{2}$  in.

**Method.**—The patient is anæsthetized and catheterized, and lies on her back, or as described under Bipolar Version. The hand is passed inside the uterus, rupturing the membranes if necessary, and the head is displaced in the direction of the child's back. A knee is sought for and grasped and brought down to the os, the fundus being steadied meanwhile by the external hand.

A. W. BOURNE.

**VERTIGO.**—The subjective state caused by an illusory sensation of displacement of one's body in relation to its environment, or of displacement of the environment in relation to one's body. Actual displacement is not in itself sufficient to produce the psychical state of giddiness. A waltzer is not necessarily giddy while waltzing, when he actually feels the rotatory displacement of his body; he may become giddy when he stops dancing, because there is then a conflict between the illusory sensation of rotation arising from the after-effect of stimulation of the semicircular canals, and the positive sensations from other

parts of his body that inform him that he is really stationary.

Knowledge of the position of one's body in space depends firstly on the sensations derived from extrinsic sources by the organs of touch and sight, and secondly on intrinsic sources of sensation—the kinæsthetic sensations from the eye and the skeletal muscles, the semicircular canals and otoliths, and visceral sensations. Any disturbance of the balance normally existing between these various groups of sensations may give rise to giddiness. Except, however, when the disturbance arises from the vestibular apparatus or the intimately connected oculo-motor system, such giddiness is not associated with any definite feeling of rotation in one direction. The patient suffers the distressing sense of a general loss of equilibrium without being conscious of a tendency to fall in any one direction. Giddiness may, in addition, be caused by any sudden alteration of cerebral blood-pressure such as results from a rapid standing up from the recumbent position, or by engorgement of the splanchnic area from shock or visceral disturbances. It is only, however, when a definite sensation of displacement of the body or of surrounding objects in one of the three directions of space is complained of that the symptoms assume diagnostic importance, and it is customary to distinguish them as vertigo in contradistinction to the ill-defined sensations of displacement which are known as giddiness.

**Vertigo due to lesions of the internal ear.**—Most forms of vertigo produced by movement—e.g. waltzing—are due to stimulation of the terminations of the vestibular nerve in the cristæ ampullæ of the semicircular canals by alterations of the pressure of the endolymph. It is therefore the vestibular apparatus that will be first suspected when the patient complains of vertigo, either as a sensation of rotation in one direction or as a sensation of falling forward or backward. Such symptoms may be found whenever the vestibule is irritated by disease of the middle ear.

The more violent attacks of vertigo associated with disease of the internal ear are usually referred to as *Menière's disease*, which is dealt with in a separate article.

**Vertigo due to lesions of the cerebellum.**—Owing to the connexion of the cerebellum with the vestibular neurones, cerebellar lesions generally give rise to vertigo with definite sensations of displacement; deafness is, however, absent. In such cases the signs



## VISCEROPTOSIS

of increased intracranial pressure will generally serve to distinguish the vertigo from that due to a vestibular lesion. Vertigo may also be a symptom of disseminated sclerosis when the cerebellum is affected.

**Tumours involving the vestibular nerve** give rise to vertigo associated with deafness and signs of increased intracranial pressure.

Vertigo from **disturbance of the ocular muscles** may be provoked by any lesion tending to cause diplopia. As a rule it is transient, the patient learning to disregard the erroneous visual localization. Vertigo is occasionally an aura of epilepsy, preceding a major or minor attack, and sometimes constituting practically the whole of a minor fit. In the latter case the non-occurrence of vomiting, the short duration of the attack, and the absence of permanent deafness or tinnitus, will enable a correct diagnosis to be made.

The vertigo of **hysteria** is rarely described by the patient as a sensation of rotation in any one direction. During an attack his gait shows the well marked characteristics of hysterical astasia-abasia, and the other symptoms characterizing Menière's disease will not be spontaneously complained of.

F. L. GOLLA.

**VESICAL CALCULUS** (*see* URINARY CALCULI).

**VESICULITIS** (*see* GONORRHOEA).

**VILLOUS ARTHRITIS** (*see* RHEUMATOID ARTHRITIS).

**VINOENT'S ANGINA** (*see* TONSILLITIS, ACUTE; DIPHTHERIA; BACTERIOLOGY AND PATHOLOGY, CLINICAL).

**VIRGINITY, SIGNS OF** (*see* RAPE).

**VISCOERAL NEURALGIA** (*see* NEURALGIA).

**VISCEROPTOSIS** (*syn.* Splanchnoptosis; Glénard's Disease).—Abnormal descent of the abdominal viscera. It is usually associated with complaints of irregular abdominal pains and dyspepsia, and many of the sufferers are distinctly neurasthenic. Objectively, there is flattening of the epigastrium with distension of the lower abdomen when the erect position is assumed.

**Etiology and symptomatology.**—The viscera are normally kept in position by the slight positive intra-abdominal pressure maintained by the tonic action of muscles of the

abdominal wall and pelvic floor. Collections of extraperitoneal fat also serve to anchor certain organs like the kidney in position. Coffey has emphasized the important part played by the forward curve of the lumbar vertebræ and the attached psoas muscles in forming a sort of shelf upon which the viscera are held by the taut muscles of the abdominal wall. Normally, the peritoneal ligaments are relaxed and appear to take no active part in maintaining organs in position, but in cases of visceroptosis they are put on the stretch by the descent of the viscera and serve as true ligaments.

Two groups of cases are recognized, both more frequent in women:

- (1) Those which depend upon defective skeletal development.
- (2) Those consequent upon a diminution of intra-abdominal pressure and a laxity of abdominal muscles.

In the first group the thorax is long and narrow, the ribs are more oblique than usual, the epigastric angle is acute, and the anterior convexity of the lumbar spine diminished. As a consequence, not only is there considerable narrowing of the upper part of the abdomen with downward displacement of viscera, but such descent is facilitated by the lack of convexity of the lumbar portion of the spine. Many of these cases are the result of unfavourable dietetic and hygienic conditions in early childhood. There may be a history of some definite disorder of nutrition such as rickets or of some disability leading to imperfect expansion of the chest (nasal obstruction, fibroid lung, etc.). The greater frequency in women is to be attributed to the restricted opportunities of girls, as compared with boys for exercise and drill.

In the second group the disability can often be definitely attributed to some depressing influence whereby muscular tone is diminished. Thus, an exhausting illness, leading not only to tenderness and wasting of the abdominal muscles but also to a diminution of intra-abdominal fat, is sometimes the starting-point. In other instances muscles fail to regain their tone after having been overstretched by a large abdominal swelling or frequent pregnancies. In either case the viscera, acted on by gravity, tend to be displaced downwards, and their peritoneal attachments, which are normally relaxed, become stretched. This stretching may possibly account for the vague abdominal discomfort and dragging pain so often complained of

## VISCEROPTOSIS

In cases of pure visceroptosis symptoms are only prominent while the patient is in the erect position, and disappear when she lies down. Temporary relief is also obtained if the lower abdomen be compressed by means of the hand or supported by a belt. Subjects of a severe grade of visceroptosis not uncommonly complain of difficulty of breathing and shortness of breath. An X-ray examination reveals the fact that in these cases the diaphragm has undergone downward displacement with consequent limitation of its excursions. As the lower abdomen is compressed, the diaphragm will be seen to rise and its normal respiratory movements once more to return. Then, too, the centre of gravity of the body tends to pass to a more anterior plane. In order to maintain the upright position of the spine, muscles and ligaments unused to strain are brought into play, and this fact may possibly serve to account for the frequent backache met with clinically. One other phenomenon may be mentioned. Abnormal rapidity of the pulse and faintness not infrequently attend the assumption of the upright position in these cases. It would appear that when the abdominal muscles have lost their tone, blood is allowed to collect within the splanchnic vessels, with the result that the heart, in order to maintain an efficient circulation, is forced to accelerate its contractions.

It sometimes happens that downward displacement of one particular organ forms the chief feature of a case. The stomach, right kidney, and transverse colon are the organs most frequently coming under observation. *Gastropotosis* may be easily recognized by radiographic examination. Pulsation of the aorta in the epigastrium is often a prominent feature of these cases. The stomach is frequently atonic, and subacidity is the rule. Kinking is more likely to occur at the junction of the first and second parts of the duodenum than at the pylorus itself. In cases of *ptosis of the colon* the transverse colon is both distended and definitely increased in length. The loosely attached hepatic flexure suffers greater displacement than the more firmly fixed splenic flexure. In extreme cases a sharp kink may occur in the latter situation, leading to difficulty in the passage of intestinal contents. Constipation is a salient symptom, and troublesome attacks of colic may occur, the pain subsiding as flatus passes. The prolapsed transverse colon is sometimes palpable, but a radioscopic examination is frequently necessary

for its recognition. The *right kidney* is more often displaced than the left. If it be entirely outside the costal arch, so that it can easily be moved to the mid-line, it is often referred to as a floating kidney, whereas the term movable kidney is applied in those cases in which, at the end of a deep inspiration, it is possible to insert the fingers above the upper pole of the organ and prevent its return. Dietl's crises and intermittent hydronephrosis are serious but infrequent consequences of renal mobility. Dietl's crises are characterized by paroxysmal attacks of pain in the region of the kidney accompanied by nausea, vomiting, and collapse. The kidney becomes tender and swollen, and is less freely movable than usual. The urine is often scanty, and may contain blood. The attack usually subsides after a few days; it is probably due to twisting or kinking of the renal vessels.

Of recent years much attention has been paid to the relation between visceroptosis and *intestinal stasis*. Some investigators incline to the view that the various bands and membranes associated with ileal and colonic stasis are to be regarded as the outcome of a physiological response to the pull of displaced organs. It must be borne in mind, however, that the membranes in question are frequently to be demonstrated in the fetus, and the exact relationship between ptosis and the presence of these various bands and membranes still awaits elucidation. In some cases it would seem that very high grades of ptosis may occur without giving rise to symptoms, but that where membranes are present and the viscera are held up at certain points, kinking of the intestine and partial obstruction arise. Some have concluded that ptosis is only likely to give rise to symptoms when it develops in one in whom bands and membranes are present.

**Treatment.**—The most important measure is to raise the intra-abdominal pressure. In severe cases complete rest in bed for several weeks is the surest means of relieving the pain and of giving the overstretched muscles a chance of recovery. The foot of the bed should be raised 6-9 in. If the digestive powers are good the diet should be generous, but it is wise to rely on small frequent meals and the taking of fluid a short time before solid food lest dilatation of the stomach be superadded. The return to normal life should be by gradual stages, and before getting up the patient should be fitted with some form

## VITAMINS

of abdominal support which will exert pressure upon the whole of the lower segment of the abdomen in an upward and backward direction.

In less severe cases the patient will be wise to lie down for an hour or two after meals. Gentle Swedish exercises systematically carried out will gradually improve the condition of the abdominal muscles. Massage, both general and abdominal, may be of service, and breathing exercises are likely to be beneficial. Tight corsets must be forbidden. Anæmia may require the use of iron or arsenic, and full pharmacopœial doses of tincture of nux vomica will help to restore tone to the muscles and improve the patients. When the neurasthenic symptoms are pronounced a modified Weir Mitchell treatment is of service.

Surgical interference is not to be recommended in cases of simple visceroptosis. It is rare for a single organ alone to be displaced, and the effect of operations undertaken to relieve pain usually passes off as soon as the patient begins to leave her bed. Shortening of the round ligaments to correct retroversion of the uterus, when retroversion is part of a general visceroptosis, is a case in point. Operation is, however, definitely indicated when prolapse of the kidney is followed by signs of obstruction to the renal circulation or to the urinary flow.

C. E. LAKIN.

**VISION, TESTS OF** (*see* EYE, EXAMINATION OF).

**VISUAL SYSTEM** (*see* NERVOUS SYSTEM, PHYSIOLOGY OF).

**VISUAL SYSTEM, LESIONS OF** (*see* NERVOUS SYSTEM, CENTRAL, LOCAL LESIONS OF).

**VITAMINS (ACCESSORY FOOD FACTORS).**—The term "vitamines" was introduced by Casimir Funk to designate certain elements of food which are of unknown composition but which are essential to growth and health. Since there is no proof that the bodies are amines, "vitamins" is a preferable spelling, or they may be termed "accessory food factors." Those which have hitherto been recognized are three in number, and are known as (1) fat-soluble A, (2) water-soluble B, and (3) water-soluble C. These bodies are present in food in only minute quantities and would appear to be nitrogenous, but their exact analysis has still to be made.

The animal kingdom is dependent upon the vegetable for its supply of vitamins.

In connexion with the study of these essential food elements the question of a proper food-balance has continually to be borne in mind. Starvation *per se* does not produce the so-called deficiency diseases—e.g. beriberi or scurvy—but the cause is a relative insufficiency of certain vitamins. Thus, simple reduction in the amount of a food poor in vitamins will lead to a disappearance of the deficiency disease, even though no increase of vitamins be made.

**Fat-soluble A** (Growth Factor) is essential to growth and is probably also necessary to well-being in adults. According to Mellanby a lack of fat-soluble A leads to the production of rickets. He has shown that puppies fed on a diet which would otherwise produce rickets may be protected by administering at the same time foods which contain this factor. Some other animals—e.g. rats—would appear to suffer from its deprivation by failure to grow, but without the development of rickets. The question whether rickets in human infants is due wholly or partially to an insufficient supply of fat-soluble A is still an open one. Xerophthalmia, a serious affection of the eye liable to cause blindness, has also been attributed to lack of this vitamin. Fat-soluble A is found in green leaves, and throughout the animal kingdom is associated with fat. There is, however, a considerable variation in the amount in different kinds of animal fat. Milk, cream, and butter, yolk of egg, beef fat, and fish oils (notably cod-liver oil and whale oil) are important sources, whilst lard (pork fat) contains relatively little, and vegetable oils, such as linseed oil, cotton-seed oil, olive oil, and palm oil, contain it in almost negligible quantity. Pea-nut oil is said to be an exception and to be rich in this constituent. It has been suggested that the milk obtained from cows fed in pastures is richer in this vitamin than that of stalled cows fed on oil cake and roots, and that thus the frequency of active rickets in the spring is explained for grass is rich in fat-soluble A, whilst roots as well as vegetable oils are deficient in it.

**Water-soluble B** (Anti-beriberi Factor, Antineuritic Factor, Growth Factor).—Deficiency in this factor is responsible for some types of beriberi, is also concerned with failure of growth in young animals, and possibly explains some of the examples of œdema with under conditions of deprivation. This

## VITAMINS

constituent is distributed widely, and exists in greatest amount in seeds and eggs. The seeds of the pulses—peas, beans, lentils, etc.—contain it throughout their substance, but in cereals, such as wheat and rice, it is confined to the germ and the layer immediately beneath the pericarp. In the milling and polishing of rice and in the milling of flour for white bread, the vitamin-containing layer and the germ are stripped off with the pericarp, and consequently a diet consisting too exclusively of white bread or of polished rice leads to the development of beriberi. Yeast and commercial yeast extract (marmite) are very rich in water-soluble B, whilst it exists also in considerable amounts in milk (but not in butter), liver, brain, pancreas, and fish roe. To a less extent it is present in many green vegetables, in potatoes, in fish, and in kidneys, but is lacking in lean meat, in fat, and in fish and vegetable oils. According to McCarrison the absence of the anti-beriberi factor leads not only to changes in the central nervous system, but to similar functional and degenerative changes in every organ and tissue in the body. The adrenal glands are hypertrophied, while other organs atrophy—notably the thymus, testicles, spleen, ovaries, pancreas, heart, liver, kidneys, stomach, thyroid, and brain, in order of severity. These changes and the consequent symptoms are brought about by nuclear starvation, in which all the nuclei of the tissue-cells share. The mixed dietary of Europeans, under ordinary conditions, serves to protect them from a lack of anti-beriberi vitamin, while its presence in milk, even in separated milk, and its comparative stability up to 120° C. explain the absence in infants of symptoms analogous to those of beriberi.

**Water-soluble C (Anti-scorbutic Factor).**—Scurvy may follow a deficiency of water-soluble C, a vitamin which corresponds to the old "vital element" of Cheadle, and is found in fresh vegetables and fruit. Oranges, lemons, cabbage, swedes, turnips, and lettuces are potent sources. Carrot juice is much inferior to lemon and orange juice, while the juice of swedes would seem to have almost equal value. Orange is ten times as protective as grape juice. Lime juice is only weakly anti-scorbutic, and the same is true of potatoes. Both milk and fresh meat contain this factor, but only in small amounts. Most authorities hold that water-soluble C is gradually destroyed at a temperature above 50° C., and rapidly above 80° C., so that boiled, condensed, or

## VITREOUS, AFFECTIONS OF

dried milks, tinned foods, and dried vegetables are deficient in this factor. American investigators, however, demonstrate that there is no diminution of water-soluble C in dried milk, and it is certainly uncommon for scurvy to occur in an infant fed in this way. The vitamin is also destroyed by alkalis, even in the cold. Bassett-Smith has shown that lemon juice may be concentrated *in vacuo* at room temperature to become a dark-yellow viscid and very acid fluid which can readily be made into tablet form with lactose, and which keeps well either as the concentrated juice or as tablets; while Harden and Zilva have demonstrated that after removal of the free acids from lemon juice the residue retains its anti-scorbutic quality. With precautions as to temperature this preparation can be concentrated and even evaporated almost to dryness without losing its potency.

Among other diseases which have recently come to be regarded as possibly due to lack of some accessory food factor are pellagra, lathyrism, and leprosy.

More work is required before we can state with precision what is the exact part played by vitamins in disease and compute the extent of food deficiency. The reaction of the human organism to lack of vitamins is far from constant, and the reason, why, under similar conditions of feeding, disease occurs in some while sparing others has still to be determined. For example, of several infants fed on a cereal proprietary food, it is only the occasional one who develops scurvy. It is probable, however, that many persons suffer from mild though abiding effects of vitamin deficiency without manifesting symptoms that lead to a diagnosis of one or other of the diseases named.

FREDERICK LANGMEAD.

## VITILIGO (see LEUCODERMIA).

**VITREOUS, AFFECTIONS OF.**—The healthy vitreous is a structure of jelly-like consistence, and is completely transparent with the exception of a few fine fibrils. The anterior layers can be examined through the dilated pupil by oblique illumination, but for a complete examination the ophthalmoscope must be used. Gross opacities can be detected by the large plane mirror used at a distance of 1 ft. from the eye, the patient being instructed to look in different directions, but finer changes require the direct method with the small mirror, and in this examination the use of a

## VITREOUS, AFFECTIONS OF

spherical +7 D lens in the aperture of the ophthalmoscope will enable the observer to focus any opacities. (If the instrument is fitted with a small plane mirror, fine opacities will be more easily seen than with the concave mirror.) Such opacities will generally appear black on the red background of the fundus; they float about freely if the vitreous is fluid, but soon come to rest if it is firm.

**Muscae volitantes** are a common phenomenon, due to the entoptic shadow on the retina of the normal vitreous fibrils. Normal persons can see them by staring at a white ground and, especially, the blue sky. If slight the phenomenon is of no significance, but when muscae are in excess they are a warning of some import and indicate a thorough examination of the globe for organic disease. The "normal" muscae are variously likened to small flies, bubbles, strings, rows of beads, etc. They differ from shadows cast by opacities in the lens or cornea in that their movements are independent to some extent of the movements of the globe; movements of the globe initiate those of the muscae, but the latter continue for a time after the former have ceased.

Pathological opacities may be formed in different ways and may be—

- (1) Degenerative,
- (2) Inflammatory,
- (3) Hæmorrhagic,
- (4) Congenital.

(1) **Degenerative opacities** are common in myopia, in which the vitreous is often extremely fluid, its jelly-like consistence being lost. The streamer-like opacities are then seen to be freely floating about, independently of the movements of the globe. If they are very abundant a detachment of the retina must carefully be looked for.

A special type is *synchysis scintillans*, in which the vitreous is beset with sparkling crystal-like bodies likened to a shower of golden rain. These are due to crystals of cholesterin, which are often the result of preceding hæmorrhage. No treatment is of value.

(2) **Inflammatory exudates** into the vitreous may form (a) very fine dustlike opacities, focused only with difficulty; (b) membranes of varying density; (c) pus. Apart from infection introduced through a perforating wound, these exudates are caused by disease of the vascular coats of the eye, especially cyclitis and choroiditis; vision is interfered with in accordance with the density and amount of

the exudate. Purulent exudate may form one of the varieties of *pseudo-glioma*, a yellowish mass being visible behind the lens; in such a case other inflammatory signs are usually present, such as circumcorneal injection and posterior synechia, which distinguish it from true glioma of the retina.

(3) **Hæmorrhage** into the vitreous may be so dense that all fundus reflex is abolished, the pupil appearing black in using the ophthalmoscope; the diagnosis can generally be confirmed by oblique illumination, which shows some red coloration behind the pupil. Short of such extensive hæmorrhage, dense black streamers which evidently consist of blood may be seen with the ophthalmoscope.

Injury is the commonest cause of vitreous hæmorrhage, but it is sometimes spontaneous, and then usually comes from a retinal vessel: in old people arterio-sclerosis is the usual antecedent. There is a type of recurring vitreous hæmorrhage in young adults which is thought by some surgeons to be tuberculous, but a simple anæmia is often the only general affection that can be detected. The possibility of intra-ocular sarcoma must be borne in mind.

The *prognosis* with regard to recovery of sight after vitreous hæmorrhage is bad. A few cases recover vision completely in the course of months, but usually membranes become organized, sometimes giving the picture of *retinitis proliferans*. *Treatment* must be on general lines.

(4) **Congenital opacities** are most commonly due to persistence of the sheath of the hyaloid artery. This vessel carries blood in foetal life, being a continuation forwards of the central artery in the optic nerve, which ends anteriorly in a vascular sheath on the posterior surface of the developing lens. All traces of it normally disappear before birth. It may remain as a filament reaching across the vitreous from the optic disc to the back of the lens, or as an anterior or posterior band attached to one of these structures, the free end moving easily in the vitreous. Its presence is consistent with good vision.

Anomalous development of the vitreous may cause dense sheets of fibrous-like tissue in the vitreous, sometimes so marked as to cause a white reflex from the pupil, and thus merit the term *pseudo-glioma*.

F. A. JULER.

**VITREOUS, EXAMINATION OF** (*see* EYE, EXAMINATION OF).

## VOCAL CORDS, PARALYSIS OF

**VOCAL CORDS, PARALYSIS OF.**—With the exception of the crico-thyroid, which receives motor fibres from the superior laryngeal, all the muscles of the larynx are supplied by the recurrent laryngeal branches of the accessory-vagus nerves. Bilateral palsy may be due to nuclear degeneration or bulbar lesions, to neuritis of the nerves, or damage of their roots by syphilitic meningitis. Unilateral palsy most commonly results from compression of one recurrent nerve by an aneurysm or tumour or by enlarged glands in the neck or mediastinum. The larynx is also involved in bulbar paralysis, and occasionally in *tabes dorsalis* and *syringomyelia*.

The larynx is concerned in phonation, in the admission of air into the trachea, and in coughing, and the symptoms of its paralysis are disturbances of these functions. They vary according to the muscle or muscles that are affected; this can be definitely determined only by laryngoscopic examination.

In total bilateral palsy the cords are moderately abducted and motionless; there is complete aphonia, the natural explosive cough is replaced by a rush of air through the glottis, and deep inspiration may be accompanied by stridor. Complete unilateral palsy may produce no symptoms, but the voice is often low-pitched and hoarse, and coughing is impaired. Abductor weakness is the most common and important of the partial palsies; even when it is bilateral the voice is little changed and coughing may be unaffected, but inspiratory difficulty and stridor are liable to occur, especially on exertion or when the larynx is congested or inflamed. Organic palsy of the adductors is rare, but hysterical aphonia is usually due to their failure to contract. Weakness of the posterior crico-arytenoid with an abductor paresis of one cord is the most common laryngeal complication of *tabes dorsalis*. The laryngeal crises of this disease may be associated or not with paralysis of certain of the muscles.

GORDON HOLMES.

**VOICE, DISORDERS OF** (*see* SPEECH, DISTURBANCES OF).

**VOLKMANN'S ISCHÆMIC CONTRACTURE.**—A paralysis and contracture of the muscles, usually affecting the forearm.

**Etiology.**—This condition is a sequela of injuries in the neighbourhood of the elbow-joint, or of fracture of the forearm. It occurs more often in children than in adults. Patho-

## VOLKMANN'S CONTRACTURE

logically, there is a coagulative change in the muscle substance, so that the muscles become hard and rigid and then contracted. Between the muscle-fibres there is an exudation, followed by a fibrosis. The muscles are not the only structures affected, as nerve lesions may be present. All these changes result from the cutting off of the blood supply to the muscles, which is brought about, as a rule, by the splints being applied too tightly.

**Symptomatology.**—The symptoms begin within a few hours of the setting of the fracture. There is great pain and sleep is impossible. The hand swells and the fingers become blue and rigidly flexed. The changes in the muscles are fully established within forty-eight hours, but they may begin as early as six hours or be delayed for six weeks.

Constitutional disturbances usually accompany the local condition. There are headache, malaise, anorexia, and a temperature rising to 102° or 101° F. On examination the swollen hand is found in a characteristic attitude; it may be in a line with the forearm, or flexed. The cyanosed fingers are extended at the metacarpo-phalangeal joints, and flexed at the interphalangeal joints. There is a loss of the power of extension of the fingers and wrist. In cases not too advanced it is possible to extend the fingers when the wrist is flexed, but with the fingers maintained in this position it is impossible to extend the hand or the forearm without a rupture of the flexor tendons. On palpation the flexor muscles are hard and inelastic; sometimes there are blebs on the skin, and perhaps sloughs which are long in healing. Nerve lesions, when they occur, are due (1) to the accident itself, (2) to direct pressure or ischaemia, or, if they occur later, (3) to constriction by the shrinking muscles. Since there is muscular paralysis in the forearm, to test the functions of the nerves it is necessary to examine the voluntary power of movement of the intrinsic muscles of the hand. Disturbances of sensation accompany the nerve lesions, and, in advanced cases, trophic changes also. The extensor muscles are never affected.

**Treatment** is very difficult, as the lesion is extremely refractory. When left untreated the contracture is progressive for about three months, and then becomes stationary. In the early stages treatment is far more easily carried out than in neglected cases, but unfortunately the nature of the condition is not recognized when treatment is most likely to be effectual, that is, within the first twenty-four hours.

## VOLKMANN'S CONTRACTURE

This is because the patient expects the fractured limb to be painful, and does not realize that the degree to which he is suffering is abnormal. The medical attendant does not see the patient, after he has applied the splints, for perhaps two or three days, by which time the damage is done. Absolutely to ensure against this serious complication of fractures it would be necessary to visit the patient every six hours; hence in all fractures in the region of the elbow-joint or of the forearm strict injunctions should be given to the patient or his guardians that the bandages must be cut should there be complaint of great pain. In this connexion it is to be remembered that, even without the application of a splint, acute flexion of the elbow—the position adopted when there is a fracture of the lower end of the humerus—may cause such circulatory interference that ischæmic paralysis ensues. Should the medical attendant be lucky enough to see the patient in the incipient stages, the correct line of treatment to adopt has been pointed out by animal experiments. All splints and bandages must be removed, and the maintenance of fragments in proper line must be a secondary consideration for the time being. The limb may be immersed for a short period in hot water. It is then raised on a pillow, and, under anaesthesia, incisions over the sites of the muscle-bellies are made through the inextensible deep fascia.

In late cases two methods of treatment have been extolled by some surgeons: (1) tendon-lengthening, (2) removal of segments from the lower thirds of the radius and ulna. These two operations bring about a temporary alleviation of the symptoms, but as the forearm grows in length the deformity recurs. The method of Sir Robert Jones gives the best permanent results. With the wrist flexed, small light zinc splints are applied to the fingers, and, the wrist remaining in a flexed position, the fingers are gradually extended. The next day, or two days later, according to the severity of the case, the angle at the metacarpo-phalangeal joint is increased, a flat malleable splint being applied to the straightened fingers and the palm. Day by day the fingers are gradually brought into line with the flexed metacarpal bones, and the wrist is subsequently extended by the same gradual method. In bringing about the extension of the flexed joints in this way the contracted fibrous tissue around the muscles is gradually stretched, as are the degenerated muscle-fibres. Those fibres

## VOLVULUS

which are unaffected—and, except in very bad cases, some of them escape—are now in a position to effect their normal movements of the hand and fingers. Instead of employing malleable metal splints, gradual extension of the contracted fingers may be brought about by the continuous traction of elastic bands which run from a dorsal splint to each contracted finger.

C. A. PANNETT.

**VOLVULUS.**—A condition in which either a coil of gut is twisted on the longitudinal axis of its mesentery so that obstruction occurs at the base of the coil, or one loop or coil of gut is entwined round another coil so that one of the portions of intestine is occluded. It is more common in people over middle age. The commonest site is the sigmoid flexure; the next most frequent is the ileo-cæcal region. Both these portions of gut usually have a fairly long mesentery which allows of torsion.

**Etiology.**—The reasons for sigmoid volvulus are fairly clear. The sigmoid loop hangs down towards the pelvis and is often loaded with solid faeces; its base of attachment is narrow, and the weight of the loop tends to lengthen the mesentery and at the same time narrow it. The gut at the base of the loop becomes a little narrowed by traction, and a certain amount of distension follows. The movements of the intestine gradually cause the loop to become twisted. Finally, the torsion totally occludes the base of the loop, the vessels in its mesentery are strangulated, and very rapid distension of the coil occurs. Local gangrene frequently develops and, later, peritonitis. Death occurs from peritonitis unless averted by operation.

In less acute cases the coil becomes gradually distended, but the vascular supply is not impaired and the signs of obstruction are unaccompanied by signs of peritonitis.

**Symptomatology.**—When the torsion of the loop is gradual or partial the symptoms are *subacute*. The patient suffers from attacks of colicky pain, distension, and constipation, which are relieved by the opening of the bowels. The attacks become more frequent, and distension of the abdomen is noticeable. Constipation becomes inveterate, and finally an acute attack of obstruction supervenes. *Acute* volvulus is notified by severe abdominal pain which is chiefly felt in the hypogastrium, rapid distension of the abdomen, and absolute constipation. If the vessels of the loop are strangled, tenderness of the abdomen and signs

## VOMITING, CYCLICAL

of peritonitis soon appear. Vomiting may occur, but is not a very noticeable feature.

In ileo-cæcal volvulus the symptoms are those of acute obstruction of the lower end of the small intestine (*see* **INTESTINAL OBSTRUCTION**), together with the presence of a rounded tympanitic swelling in the right lumbar or epigastric region. The swelling corresponds with the distended cæcum.

**Diagnosis.**—The condition is not always diagnosed before operation. If, however, complete constipation is present (tested by two turpentine enemata) and the abdomen rapidly becomes distended and tender in a patient past middle age, one should suspect volvulus of the sigmoid.

**Treatment.**—So soon as a volvulus is diagnosed or suspected, the surgeon should be called in, since the only method of treatment is operative.

ZACHARY COPE.

**VOMIT, ANALYSIS OF** (*see* **GASTRIC CONTENTS, EXAMINATION OF**).

**VOMITING, CENTRAL** (*see* **STOMACH, FUNCTIONAL DISORDERS OF**).

**VOMITING, CYCLICAL** (*syn.* Recurrent Vomiting; Periodic Vomiting).—A form of vomiting in children, recurring at irregular intervals, accompanied by marked prostration and wasting and by the symptoms of acid-intoxication.

Although formerly little recognized, it is a condition by no means uncommon, and is of considerable clinical importance.

**Etiology.**—The actual pathogenesis is unknown; at first ascribed to acid-intoxication, cyclical vomiting is now recognized as being due to a toxæmia of more complex origin. Possibly defective functioning of the liver, either congenital or acquired, plays the chief rôle. The condition occurs more commonly in girls than in boys, and particularly between the ages of 5 and 12. A history of gout in the family is not infrequently forthcoming; in other cases there is a distinct neuropathic inheritance. Though no particular article of food and no indiscretion in diet appears culpable, yet the digestion of these children is easily upset. Fat is the form of food which is usually most abhorrent to them and the most likely to cause disturbance.

**Pathology.**—The most striking morbid change is extreme fatty degeneration of the liver, best marked in the periphery of the lobules. The heart, kidneys, and muscles

show a similar degeneration, but in a minor degree. Slight lesions amounting to follicular ulceration have been noted in the intestine, but the chief feature is the absence of a recognizable sufficient cause, unless the liver degeneration be held to be such.

**Symptomatology.**—An attack is ushered in by a few days or hours of malaise, drowsiness, headache, want of appetite, and perhaps fever. The fever is not usually high, but has been noted as high as 104° F. Examination of the urine at this stage will reveal the presence of acetone and diacetic acid in considerable quantity. The breath is peculiarly sweet and offensive from the presence of acetone. The tongue is coated, and the child will take nothing but water. Constipation is the rule, though occasionally there is diarrhœa with some abdominal pain. With the onset of vomiting the child obtains no relief, but is increasingly distressed by the forcibleness of the retching. At first food, then bile, and later perhaps some altered blood in the form of "coffee grounds," are added to the vomitus, which has the sickly sweet acetone odour. The vomiting is repeated frequently, and is evoked by the slightest suggestion of food. Thirst and a dry tongue follow, and the drowsiness increases until, in severe cases, it may lead to stupor or even to fatal coma. Jaundice and cyanosis may be present. Acid-intoxication is recognizable in this drowsiness, in air-hunger (the breathing being rapid and deep), and in the odour of the breath and the urinary changes. Irritability and restlessness, accompanied by sighing, may be prominent symptoms, and the clinical picture of meningitis may be simulated.

**Diagnosis.**—Although commonly called "*bilious attacks*," cyclical vomiting differs from what is most accurately implied by that term in the absence of a sufficient dietetic error and in the occasional fatal issue. From the periodical *nervous vomiting* of children it is distinguished by the obvious distress, the prodromal symptoms, the urinary changes, and the odour of the breath. Nervous vomiting in children, though it may lead to wasting, is gratifying rather than distressing to the patient and is not accompanied by signs of toxæmia. From *meningitis*, cyclical vomiting is diagnosed by the absence of rigidity or paralysis, and the prominence of the acid-intoxication syndrome; in early comatose cases lumbar puncture will exclude meningitis. Unnecessary deaths have been caused by confusion with *acute intestinal obstruction*. These could have been avoided



## VOMITING, CYCLICAL

had the acid-intoxication been noticed and the urine examined when no local signs, either abdominal or per rectum, were detected.

**Prognosis.**—The attack generally persists for only a few days, and is followed by almost sudden recovery; appetite, health, and inclination to play rapidly returning. Occasionally the vomiting continues for a few weeks and leads to great wasting, even to emaciation, but terminates in a similar fashion. Rarely the toxic symptoms may culminate in death. Attacks may occur as frequently as every few weeks, or may be separated by intervals of several months. Not uncommonly they cease at puberty, but may be replaced by attacks of migraine.

**Treatment.**—Although definite success cannot be promised, attacks may sometimes be prevented or reduced by careful dieting and regulation of the bowels. No special dietary is required, but fats should be limited, and the food should be plain and digestible and given regularly. Rich gravies or soups, sweets, cakes (unless very plain), uncooked fruits (especially bananas), pastry, and new bread should be avoided. No attempt should be made to restore the loss of weight rapidly by giving cod-liver oil between the attacks, or forcing the appetite by large quantities of food or delicacies. A free daily evacuation should be ensured by some simple purgative such as syrup of figs, or fresh infusion of senna pods given overnight, or citrate of magnesia in the morning. An alkaline mixture such as the following is sometimes beneficial :—

R̄ Pot. bicarb. gr. v.  
Pot. citrat. gr. v.  
Tr. nuc. vom. ℥ii.  
Inf. gent. co. ad ℥i.  
T.d.s., a.c.

For a child at. 5.

With the onset of an attack, calomel (3 gr.) or castor oil ( $\frac{1}{2}$  oz.) should be given immediately, and the child should be put to bed and allowed only milk and soda-water. Often feeding soon becomes impossible, but thirst may be allayed by giving freely of hot water; even though this is often returned, it serves a useful purpose by washing out the stomach. Large doses of alkalis such as sodium citrate or sodium bicarbonate (20 gr. hourly) are recommended, but unfortunately are seldom retained. The right procedure in severe cases is to give the bicarbonate intravenously in a 4-per-cent. solution. In urgent cases dextrose (2½ per cent. in normal saline) may be given intravenously. Alter-

## VOMITING, PERNICIOUS

natively, a 10-per-cent. solution of bicarbonate of soda in a 10-per-cent. solution of dextrose may be given per rectum. A 2-per-cent. solution of bicarbonate with a 5-per-cent. solution of dextrose may be injected subcutaneously.

FREDERICK LANGMEAD.

**VOMITING, HYSTERICAL** (*see* STOMACH, FUNCTIONAL DISORDERS OF).

**VOMITING, PERNICIOUS, OF PREGNANCY.**—True pernicious vomiting is a rare complication of pregnancy. Cases in which vomiting is excessive are fairly common, and are either of hysterical or of reflex origin, but pernicious vomiting is of toxæmic origin and is a totally different condition. It is not infrequently fatal unless the uterus is emptied in time, whereas hysterical or reflex vomiting is never fatal or really serious. The toxæmic variety occurs rather later in pregnancy, at the third or fourth month, and is accompanied by symptoms which show its toxæmic origin. The nature of the toxæmia is much the same as that of eclampsia, and must be of maternal or of fetal origin, most probably the latter in the first place (*see* PUERPERAL ECLAMPSIA). It is shown by the general condition of the patient, and particularly by the urinary secretion. The vomiting itself is extreme, everything, even water, being rejected by the stomach. The result is rapid emaciation. The urinary secretion gradually fails in quantity, whilst analysis shows the presence of albumin, casts, acetone, diacetic acid, and  $\beta$ -oxybutyric acid. The presence of acetone, etc., may be chiefly the result of starvation, but it cannot be denied that it is also the outward expression of a toxæmic process. The ammonia coefficient of nitrogen excretion is much increased—that is to say, the ratio of nitrogen excreted as urea and that excreted as ammonia reaches a figure much higher than normal. This, again, may be the effect of starvation, but it serves as an index of the serious condition of the patient. The pulse-rate and the temperature rise as in the toxæmia of eclampsia, and that there is some profound effect upon the liver is shown by the presence of jaundice in most cases. The patient gradually becomes drowsy, the urine diminishes and may be suppressed, and finally death occurs from exhaustion.

In simple excessive vomiting of hysterical or reflex origin there are none of these symptoms; the urine is normal, as also are the temperature and the pulse, the amount of weight lost

## VOMITING, POSTANÆSTHETIC

is not great, and a cure can usually be effected by isolation of the patient, abstention from mouth-feeding, and suggestion.

**Treatment.**—In toxæmic cases no treatment is of the slightest use, except emptying the uterus. The great essential is to do this before the patient is too exhausted to bear the operation and the shock attending it. When the urinary findings and the patient's general condition show that the vomiting is toxæmic, abortion should be induced at once without attempting any other form of treatment. The best way to induce abortion at the third or fourth month is to dilate the cervix with metal dilators sufficiently to allow of the introduction of a small Horrocks bag tied to a No. 12 railway catheter. If the membranes can be ruptured before putting in the bag, so much the better. The uterus should be encouraged to empty itself, but it is often inert in these cases. If abortion does not occur in a reasonable time—say twenty-four hours—an anæsthetic (not chloroform) must be given and the uterus must be cleared out with the finger and ovum forceps; a proceeding often attended by very severe hæmorrhage from the placental site. Quickness, therefore, is essential, followed by rapid plugging of the uterus as soon as emptied. Afterwards endeavour must be made to re-establish the urinary secretion and to encourage the bowels and skin to act, so that the poison may be eliminated by as many channels as possible.

THOS. G. STEVENS.

**VOMITING, POSTANÆSTHETIC** (*syn.* Delayed Chloroform Poisoning).—An unexplained toxæmia following anæsthesia, and accompanied by symptoms of acid-intoxication.

**Etiology.**—Although it occurs most commonly after chloroform anæsthesia, it is in no sense a simple poisoning by that drug, for it sometimes follows the administration of ether and even of nitrous oxide. It is almost confined to children. Neither the length or severity of the operation nor the amount of anæsthetic appears to determine its onset. Often the operation has been a short and simple one in an apparently healthy and well-nourished child. The previous history of severe "bilious attacks," and the similarity of the symptoms and post-mortem appearances to those met with in cyclical vomiting, point to a like cause underlying the two conditions. The rôle of the anæsthetic seems to be to determine and accentuate an attack.

**Pathology.**—The changes found differ only

in degree from those in cyclical vomiting. The destruction of the liver is greater, and necrotic changes in the liver-cells are added to the fatty degeneration.

**Symptoms.**—The symptoms are those of cyclical vomiting (*see* VOMITING, CYCLICAL), but are generally more severe. They may begin soon after the operation, so that the drowsiness and vomiting are insensibly merged with the usual effects of recovery from an anæsthetic, or they may be postponed for a few days. The vomiting is usually insistent, and the toxæmia great. Jaundice is almost invariable, and restlessness is very much in evidence, the child continually tossing about and sighing or crying out.

**Prognosis.**—Death is much more frequent than in cyclical vomiting, and is the usual issue. Sometimes the symptoms cease abruptly and are followed by rapid recovery.

**Treatment.**—This serious condition is largely avoidable if the following rules be adhered to:

1. In all operations on children make a careful inquiry for a history of previous "bilious attacks."

2. When such is obtained do not give chloroform.

3. Whenever possible, except in emergency operations, admit the child to a nursing home or hospital for a few days before the operation is performed, to accustom him to his surroundings and to the altered diet, and to avoid fright.

4. Examine the urine for acetone, and if this is found postpone the operation until it disappears as tested by ordinary means.

5. Avoid a period of starvation.

6. If a history of "bilious attacks" is elicited, give bicarbonate of soda (30 gr. daily) for three days, and plenty of sugar. A lump of sugar may be given immediately before the operation.

When postanæsthetic vomiting occurs its treatment is that of cyclical vomiting (*see* VOMITING, CYCLICAL).

FREDERICK LANGMEAD.

**VOMITING, REFLEX** (*see* STOMACH, FUNCTIONAL DISORDERS OF).

**VOMITING, TOXIC** (*see* STOMACH, FUNCTIONAL DISORDERS OF).

**VON JAKSCH'S ANÆMIA** (*see* ANÆMIA).

**VON PIRQUET'S REACTION** (*see* SEROLOGICAL DIAGNOSIS).

## VULVA, DISEASES OF

### VON REOKLINGHAUSEN'S DISEASE (see SKIN, FIBROMATA OF).

**VULVA, DISEASES OF.**—Under this title the following subjects are considered:—

1. MALFORMATIONS.
2. ACUTE VULVITIS.
3. CHRONIC VULVITIS.
4. KRAUROSIS VULVÆ.
5. ESTHIOMÈNE.
6. HERPES OF THE VULVA.
7. ECZEMA OF THE VULVA.
8. PRURITUS VULVÆ.
9. VASCULAR CONDITIONS OF THE VULVA.
10. CYSTS OF THE VULVA.
11. DISEASES OF THE VULVO-VAGINAL (BARTHOLIN'S) GLAND.
12. DISEASES OF THE HYMEN.
13. NEW GROWTHS OF THE VULVA.

#### 1. MALFORMATIONS

**Absence** of the vulva is decidedly rare and is associated with congenital absence of the vagina, uterus, and ovaries. Absence of one or more of the constituent parts is, though rare, more frequently seen, and may be associated with perfect internal generative organs.

A **double condition of the vulva** has been recorded.

**Congenital atresia** arises from failure of the uro-genital sinus to open externally, and consequently is associated with retention of urine and feces.

An **infantile type of vulva**—most frequently seen in cretins and dwarfs—may be maintained throughout life.

The **clitoris** may be **absent, bifid, atrophic, or hypertrophied**. The size varies within wide limits. If much enlarged, it may be subject to chafing and irritation and cause annoyance. The application of a lead-and-opium lotion or of zinc ointment, or simple bathing with a mild antiseptic, gives relief. For any considerable hypertrophy a simple amputation of the clitoris is satisfactory.

**Epispadias** and **hypospadias** are occasionally seen, and require some form of plastic operation.

**Anomalies of the hymen** are sometimes encountered; thus an imperforate hymen, giving rise to retention of menses, is not uncommon (see AMENORRHOEA), and in some cases of double vagina a double hymen has been recorded.

#### 2. ACUTE VULVITIS

The classical specific forms, such as the erysipelatous, diphtheritic, and gangrenous,

have, under conditions of modern hygiene and nourishment, almost disappeared, leaving only, among the acute inflammations, a mild pyogenic form chiefly following the injuries incidental to childbirth in adults, and among the specific the gonococcal infection.

**Acute septic vulvitis** occurs both in children and in adults. In the former it results from perineal irritation, scratching, and infection; in the latter it is most commonly seen during the puerperium, arising and extending from the perineal and vaginal tears or the abrasions of the labia so frequently encountered. It sometimes results from the use of strong antiseptic solutions or of excessively hot douches, and the application of the ordinary solution of iodine in spirit.

In the early stages there is severe burning pain. This is soon followed by cedema, redness, and swelling, and later by an abundant secretion which, starting as a serous effusion, soon becomes purulent.

**Treatment.**—The use of lint wrung out of hot lead-and-opium lotion or eusol solution may be advised. As the secretion forms, a sitz-bath twice daily is comforting and aids recovery, fomentations being still frequently applied. In every case the labia should be separated carefully and gently swabbed two or three times a day with a mild antiseptic solution, an ointment such as the yellow oxide of mercury being applied immediately afterwards to prevent adhesions and assist healing.

**Gonococcal vulvitis** can only be recognized with certainty by examining the discharge under the microscope, but may be suspected if the inflammation involves the urethra as well as the vulva and is accompanied by much swelling and a free greenish discharge. Involvement of Bartholin's gland is held to be typical, but many of the symptoms of gonorrhœal vulvitis are due to a mixed infection. The *treatment* is similar to that for acute purulent vulvitis, except that, in addition, particular care should be taken to avoid extension of the infection to the vagina and cervix.

**Membranous vulvitis** is characterized by a greyish membrane which covers the whole or a part of the vulva and, when stripped off, leaves a bleeding granular surface. It used to be seen after childbirth, and in some cases the Klebs-Löffler bacillus has been cultivated. If encountered, it should be treated by the appropriate serum.

An **erysipelatous inflammation** was also not

## VULVA, DISEASES OF

infrequently seen after septic labours, but is now rare. It can be identified by a reddish induration of the labium, showing a margin of advancing inflammation, and by a high temperature, with perhaps delirium. The *treatment* adopted is the application of hot fomentations and the administration of liq. ferri perchloridi.

A **gangrenous** state of the vulva is very rare, but has occurred as the result of extensive injury in delivery and in acute specific fevers. Debilitated children, the subjects of an acute exanthematous fever, are liable to a special variety known as **noma vulvæ**, which is usually rapidly fatal. One labium becomes swollen and black, and sloughs form. The condition calls for immediate resection of the affected labium.

### 3. CHRONIC VULVITIS

**Simple chronic vulvitis** is usually the result of an acute condition in which recovery has been incomplete. In one of the folds between the labia an area with roughened surface and secreting pus is found. The discharge may be the only symptom. Treatment consists in careful swabbing with a mild antiseptic and the application of gauze saturated with red lotion.

**Leukoplakic vulvitis** is a form of chronic vulvitis of long standing, and is identified by characteristic changes in the skin of the vulva. Areas of whitened and sodden epithelium are seen, with perhaps fissures at other points. It appears to result from persistent irritation of the part from some trivial and intractable cause such as those noted below under Pruritus Vulvæ. The changes may affect the whole or only a part of the vulva and extend to the thighs and perianal region.

In the early stages the affected part is a little reddened, perhaps swollen, excoriated, roughened, and dry; later, with persistent chronic inflammation of the corium, the skin over the affected areas becomes whitened. When desquamation is slow it is thickened and sodden in some areas; whilst others show a thin, dry, white skin, indicating considerable fibrosis of the tissues beneath the epidermis. In the later stages these areas of thin white skin, owing to the contraction of the deep-seated fibrous tissue, develop small fissures which, persisting for some time, become ulcers with malignant characters. Some atrophy of the labia is associated with the chronic changes.

Microscopically, there are small inflammatory areas in the subepithelial tissue immediately beneath the corium. The consequence is a

fibrotic change which interferes with the vitality and ordered growth of the epithelium.

The dominant symptom is a persistent irritation which exacerbates when the patient is in bed at night, and leads to scratching until soreness is produced. Any fissures that develop are painful and tender to the touch, and the irritation and the tenderness in the excoriated parts are sufficient to render life a burden. The patients are all between 40 and 60 years of age.

*Treatment.*—In a definite case little time need be lost in palliative treatment, which consists in the application of lead lotion at night and of a dusting powder during the day. Morning and evening the vulva should be carefully washed and dried.

When the disease is well developed, the best treatment is excision of the vulva or, if it is localized, of the part affected. The presence of fissures denotes a precancerous state and calls for wide resection.

### 4. KRAUROSIS VULVÆ

A rare form of atrophy of the vulva with stenosis of the vaginal outlet. It occurs after the menopause, whether natural or artificial, and, very rarely, in younger people the subjects of sterility. The symptoms are pain, soreness, and great sensitiveness in the vulva. There is dyspareunia and, occasionally, painful micturition. The labia minora and introitus are atrophied conspicuously, but the labia majora no more than is commensurate with the age-period. The application of sedative lotions and ointments gives relief to the symptoms, which, except the dyspareunia, eventually subside.

### 5. ESTHIOMÈNE

This is characterized by the formation of a large firm swelling occupying the labium minus or clitoris, and liable to ulceration. The indurated mass is sessile and is a variety of granulomatous fibrous swelling, probably of syphilitic nature; in any suspected case a Wassermann reaction should be tried. The affection is rare, and is treated by antisyphilitic remedies.

### 6. HERPES VULVÆ

A typical chain of herpetic vesicles which has been preceded by severe neuralgic pain in one labium majus or in the mons veneris is sometimes encountered. The vesicles soon dry with cleanliness and the application of a dusting powder, but an irritation remains for a few weeks.

## VULVA, DISEASES OF

### 7. ECZEMA OF THE VULVA

Ecze<sup>m</sup>a sometimes occurs on the vulva as well as in the groin and thighs; it is usually of the weeping variety and reacts to ung. plumbi or calamine lotion.

### 8. PRURITUS VULVÆ

This condition is considered in the article PRURITUS, but here it may be added that a particularly disagreeable form is that known as **pruritus of pregnancy**, which may start from the third to the seventh month of pregnancy and persist to its end. It is toxæmic in origin, and ceases immediately at the termination of pregnancy. The itching is excessively aggravating, rendering life almost unbearable. The vulva shows no characteristic changes, and but little relief is obtained from local applications. To afford relief it may, in the worst cases, be necessary to empty the uterus after the child is viable.

### 9. VASCULAR CONDITIONS OF THE VULVA

**Varicose veins** are generally seen during pregnancy, but they also occur with a pelvic tumour or displacement of long standing. The appearance is that of elongated, irregular, knotty masses of a bluish colour in one or both labia. If greatly enlarged, as is frequently the case in pregnancy, they cause a feeling of heaviness in the vulva, soon followed by irritation and a variable amount of discomfort. When due to pregnancy they diminish considerably in size after childbirth, but with each subsequent pregnancy become more evident and tend to persist.

Liability to injury, with subsequent hæmorrhage or thrombosis, is the only danger. The bleeding which takes place may be external and free, when it may even be alarming, but is temporarily controlled by direct pressure; or a hæmatoma of great size may rapidly form. Thrombosis sometimes occurs during the puerperium.

**Treatment.**—Whenever large or causing inconvenience the veins are best treated by excision. Amelioration during pregnancy is obtained by resting in a recumbent position with the pelvis raised, and by wearing a vulval pad under a tightly drawn T-bandage. Compresses of spirit lotion or lead and opium may relieve the itching and burning.

**Oedema of the vulva** occurs to a marked degree and is seen at its worst in some cases of albuminuria of pregnancy with general anasarca. In a milder form it is met with

in uncompensated heart lesions, cirrhosis of the liver, large pelvic tumours, and certain renal conditions. The labia are swollen, white, often vesicular, and may attain a very large size. Treatment should be directed to the cause. Punctures of the labia are seldom necessary, and should be avoided, if possible, in the albuminuria of pregnancy, from fear of the risk of subsequent infection.

**Hæmatoma vulvæ** follows rupture of a vessel, commonly a varicose vein, as a result of some injury. A rapidly-growing, very tender swelling appears in one labium and may extend widely, infiltrating the perineum and the abdominal fascial plane, and occasionally spreading upwards along the planes of pelvic fascia to form a lateral vaginal mass. The history, discoloration of the skin, and presence of varicose veins indicate the nature of the swelling. Unless small, a hæmatoma requires aseptic evacuation of the clot and control of the bleeding. Temporary relief from pain may be afforded by the application of spirit lotions.

**Elephantiasis of the vulva** is considered in the article on FILARIASIS.

### 10. CYSTS OF THE VULVA

Of the cystic swellings of the vulva, the great majority arise in connexion with Bartholin's gland (*see below*). Less commonly, small sebaceous cysts, small cysts of the hymen, and a hydrocele of the canal of Nuck are met with.

A **hydrocele of the canal of Nuck** is a collection of fluid in the vaginal process of peritoneum ensheathing the round ligament of the uterus. It may be encysted and loculated, and presents itself as a fluctuating, painless swelling over the spine of the pubis, perhaps extending into the anterior end of the labium majus. It must be distinguished from an inguinal hernia, and very rarely may be mistaken for a cyst of Bartholin's gland. The encysted fluid may be removed.

**Sebaceous cysts** form small yellowish spherical flat swellings, either on the labia majora or the pubes. They are seldom noted until, owing to infection, they become painful and suppurate. If evacuated by pressure they seldom recur.

### 11. DISEASES OF THE VULVO-VAGINAL (BARTHOLIN'S) GLAND

Diseases of Bartholin's gland include cysts of the gland or the duct, inflammation and

## VULVA, DISEASES OF

abscess of the gland, and new growths such as adenoma and adeno-carcinoma.

**Cysts of the duct** are due to stenosis from an earlier inflammation. The swelling is superficial and situated beneath the stretched skin of the labium majus. It is thin-walled, ovoid, seldom exceeds the size of an egg, and encroaches upon the vaginal outlet. The cyst contains clear watery fluid unless it has been injured, and microscopically the lining consists of flattened cubical cells. The gland itself is found somewhat flattened out on the deep surface of the cyst.

**Cysts of the gland** form less prominent and rather ill-defined swellings, lying deeply in the posterior part of the labium majus, where they cause less deformity. One finger passed into the vaginal introitus and a second placed externally usually serve to define the shape and size of the swelling, which may be multilocular and irregular in outline, or single and spherical. The contents of the cyst are usually mucoid, and it is lined with columnar epithelium; when multilocular its features, as seen under the microscope, resemble those of a cystic adenoma in some cases. The symptoms are those of an inconvenient swelling in the vulva, prone to become chafed, inflamed, and even to suppurate. It is not usually painful or sensitive.

The *diagnosis* of Bartholinian swellings is not difficult. Their site and size serve to distinguish them from other cystic conditions.

The *treatment* is excision. An oval area of stretched skin on the surface of the cyst and the gland on the deep surface must also be removed.

**Inflammation** of Bartholin's gland is encountered in the course of an acute or a chronic vulvitis, and frequently muco-pus can be made to exude from the duct, especially in gonorrhoeal infections which have apparently cleared up. The gland, when indurated and enlarged, can be palpated, lying deeply in the posterior part of the labium majus. The integument over it, and particularly round the mouth of the duct, where it opens just behind the hymen, is red, injected, and inflamed.

An **abscess** forms an indurated swelling with a fluctuating centre in the posterior half of the labium majus and the labium minus, its size and contour depending upon the amount of cellulitis in the neighbouring tissues. Spontaneous rupture, with escape of contents, results in a sinus and recurrent abscesses. The symptoms are pain, swelling, and redness of

part of the vulva. When the abscess has formed, the pain is very acute, the temperature is raised, and tenderness to touch is very evident. *Treatment* consists in freely opening and packing to secure healing from the depths. In cases which have recurred, either from a former spontaneous rupture or from an insufficiently free incision, it is better to operate in a quiescent period and excise the whole gland.

**New growths** of the gland are very rare. Adeno-carcinomata and endotheliomata have both been recorded, the former being the more common.

### 12. DISEASES OF THE HYMEN

Minute cysts have frequently been noted on or near the free margin of the hymen, and are often multiple. They are thought to be congenital in origin, and are usually lined with cylindrical but occasionally with stratified epithelium.

**Inflammation** results from laceration and subsequent infection of the abraded areas. On examination, one or more tender points sensitive to the touch are found. This condition, it is estimated, accounts for 50 per cent. of cases of dyspareunia; the sensitiveness excites a reflex spasmodic contraction in the sphincter vaginae which often leads to the mistaken diagnosis of vaginismus. Careful excision of the hymen, with accurate suture of the vulvo-vaginal margins, is invariably successful, provided that a sufficiently long convalescence is insisted upon, so that healing is perfect before the risk of further trauma is permitted.

### 13. NEW GROWTHS OF THE VULVA

#### (1) SIMPLE NEW GROWTHS

A **fibroma** is the commonest of the simple growths. It arises in the subcutaneous connective tissues of the labium majus or minus. In its early stages it forms a smooth, hard, sessile growth of spherical or oval form, but, owing to the laxity of the tissues in which it arises, it soon becomes pedunculated and pendulous. Myxomatous degeneration sometimes occurs in the tumour, causing it to become cystic. The tumour is excised without difficulty after clamping and tying the vessels in the pedicle.

A **lipoma** is occasionally found as a more or less circumscribed soft swelling situated in one of the labia, with puckering of the skin over it. The only symptoms are those attributable

## VULVA, DISEASES OF

to the inconvenience of the tumour. Treatment is by enucleation.

**Papillomata.**—A true papilloma is rare. It has to be distinguished from a squamous-celled carcinoma, which may start as a small papillary growth; and from a venereal wart, which, however, is seldom solitary.

Multiple papillomata are of venereal origin. They are recognized by the area of infection, often extending to the perineum and the thighs, the moisture of the opposed surfaces, and a peculiar offensive odour. If discrete and small, they may be treated by cleansing with ordinary antiseptic solutions and dusting with calomel powder. The larger growths should be cut away and their bases cauterized with the thermo-cautery.

### (2) MALIGNANT NEW GROWTHS

The vulva is not particularly prone to malignant disease, the proportion of growths arising in this part, as compared with female genital cancers, being 1 in 40. Much the commonest form is an epithelioma. Sarcomata, adeno-carcinomata, and endotheliomata also occur.

**Epithelioma, or squamous-celled carcinoma.**—The onset is usually in later life, 75 per cent. of the cases arising between 40 and 60 years of age, and 20 per cent. after 60 years. Its site may be any part of the vulva, most commonly the inner or outer surface of the labium majus, less frequently the frænum clitoridis, the fourchette, and the anterior and posterior commissures. Preceding the growth there is usually a state of chronic irritation of long duration.

The growth may start as a small, hard, circumscribed nodule with either a smooth or a papillary surface. Disintegration usually occurs before any great size is attained, and a small, dirty, elevated ulcer with everted edges results. The base of the ulcer is irregular, bleeds freely when rubbed, and is covered with purulent secretion. In advanced cases the growth may form deep crater-like elevations with irregular elevated edges.

Involvement of the inguinal glands is early, first on the affected but very soon also on the opposite side. The growth itself may extend to the vagina, and so to the rectum and bladder. Occasionally a contact cancer is noted on the opposite labium.

The initial symptom may be discharge perhaps bloodstained, from an early ulceration. In some cases pain is the first indication and in others the presence of a small nodule. In the later stages the distress caused by a foul ulcerating mass, ready to bleed on the slightest provocation, is extreme, and to this may be added a severe type of pain as the growth extends to the deeper tissues.

In making a *diagnosis*, the age and any evidence of antecedent irritation, especially of leukoplakia, should be taken into account. When in the case of a patient over 40 a nodule or small ulcer or fissure in the vulva is found, the possibility of a syphilitic origin should be excluded by the Wassermann test; if then the nature of the growth is not evident, a piece should be removed for microscopical examination.

The *prognosis* is not good, but early diagnosis is capable of greatly altering this outlook. The factors in favour of recurrence are late diagnosis, advanced age of the patient, early lymph-gland infection, and finally proximity to the pubic arch and urethra, which favour incomplete removal. The cures in cancer of the vulva are estimated at 15 per cent., as compared with 25 per cent. for cancer of the cervix and 75 per cent. for cancer of the corpus.

*Treatment* consists in early and radical removal of the growth together with the lymphatic areas in one mass.

**Cancer of Bartholin's gland** is rare, but may take the form either of a malignant adenoma or, much less commonly, of an endothelioma.

**Sarcoma of the vulva** is infrequent, but may occur either as the spindle- or the round-celled variety. More common is the so-called **melanotic sarcoma**, which may appear in any part of the vulva but is most often seen in the neighbourhood of the urethra. In character it is a small, pigmented, almost black mass, and is of recent origin.

The only growth for which it can be mistaken is a metastatic form of syncytioma, but a history of a recent pregnancy terminating in a hydatidiform mole and rapid onset of bleeding at the site of the growth serve to distinguish it from the latter.

BRYDEN GLENDINING.

**VULVO - VAGINITIS IN CHILDREN**  
(see VAGINITIS).

## WARTS

**WAR NEPHRITIS** (see NEPHRITIS).

**WAR NEUROSES** (see NEUROSES, WAR).

**WAR OEDEMA** (see OEDEMA).

**WARTS.**—The common wart (*verruca vulgaris*) is a benign papillary excrescence of a horny, friable character.

**Etiology.**—It is probable that just as there are several clinical varieties of the condition, there is more than one etiological factor. That certain types are contagious has been amply demonstrated by Jadassohn and others, who inoculated themselves and their pupils with emulsions made from excised specimens and, after a more or less prolonged incubation period, produced crops of warts which were identical with the original. The virus has not been isolated.

**Histopathology.**—Sections of warts show them to be true papillomata consisting of protuberant groups of Malpighian papillæ complete in every detail with nerves and blood-vessels and protected superficially by marked hypertrophy of the corneous layers.

**Symptomatology.**—Some warts grow on a flat base, others exhibit a tendency to pedunculation, and I have noticed that the latter is the rule for warts appearing on the scalps of young adults with seborrhœic tendencies. The *senile wart* (*verruca plana senilis*) appears on the trunk mainly, and on the limbs of old and generally seborrhœic subjects, as well-defined flat elevations of a papillomatous type, usually covered by dry, brownish crusts of greasy scales, which on removal with benzine expose the soft papillary structure underlying them. The commonest type of wart is the *verruca juvenilis* of young women and children. For purposes of classification it may be divided into a soft, contagious variety, appearing in crops of pearly papules on the face, neck, backs of the hands, and the much harder and superficially friable type which tends to occur on the knuckles of the fingers and bony prominences of the hands, and when it occurs in the nail-fold may give rise to considerable discomfort, and difficulty in treatment.

**Diagnosis.**—On superficial, careless examination the papular contagious type of wart is not unlike *molluscum contagiosum*. The latter can, however, readily be distinguished by its central dell or depression, and easily expressible

contents. Warty moles and *navi* are usually pigmented and date from birth. Large warts on the hands and knees and warty growths elsewhere should always arouse the suspicion of an underlying *lupus verrucosus*.

**Treatment.**—In spite of the many scientific and quack remedies, ranging from radium to incantations, which have been and continue to be vaunted for the cure of the disfigurement, we are still without a specific for it. In utilizing one or more of the many agents that are recommended, one should avoid those which produce scars, e.g. the fuming acids which penetrate sufficiently to destroy the Malpighian body. The judicious application of a 20-per-cent. salicylic-acid plaster, to be renewed every twenty-four hours, though a tedious is a painless and usually successful method. If a liquid caustic is used, pure acetic or trichloroacetic acid is the best. The remedy is applied on a pointed match, and the surrounding skin ought to be protected with vaselin. For the face a solution of salicylic and lactic acids in flexile collodion, separately applied to each wart, is a valuable method. Great success is claimed by many authors for CO<sub>2</sub> snow, but I have found it by no means certain, very often painful, and open to the criticism that its action is not sufficiently local. I have seen unnecessary scarring with this procedure. For general purposes the light application of a Paquelin or galvanocautery, the rapid action of which is absolutely local and almost painless at a dull-red heat, has given me the best results. Children must usually be anesthetized.

Among the best modern methods of treatment, of the multiple juvenile type at any rate, are radium and X-rays. The latter should be given in small doses of  $\frac{1}{4}$  pastille at a sitting, and the standard pastille dose ought not to be exceeded for so trivial an affection. Ionization with zinc sulphate is an efficient but painful method. For the small and obviously infective warts on the faces of children I have found exfoliation by a five-minutes' exposure at 25 cm. to the Kromayer lamp an efficient and rapid cure. Great care must be taken to shield the eyes of patient and operator from the dangerous effects of the ultra-violet rays on the retina.

HENRY SEMON.

**WARTS, POST-MORTEM** (see SKIN, TUBERCULOSIS OF).



## WHITLOW AND INFECTIONS OF THE HAND

**WASSERMANN REACTION** (*see* SEROLOGICAL DIAGNOSIS).

**WATERBRASH** (*see* STOMACH, FUNCTIONAL DISORDERS OF).

**WEIL'S DISEASE** (*see* Spirochæstosis Ictero-hæmorrhagica, under JAUNDICE).

**WEIR-MITCHELL TREATMENT** (*see* NEURASTHENIA).

**WENS** (*see* SEBACEOUS CYSTS).

**WERLHOF'S DISEASE** (*see* PURPURA).

**WHIPWORM** (*see* INTESTINAL WORMS).

**WHITE KIDNEY, CONTRACTED** (*see* NEPHRITIS).

**WHITE LEG** (*see* PHLEGMASIA ALBA DOLENS).

**WHITE PNEUMONIA** (*see* LUNG, SYPHILIS OF).

**"WHITE-SPOT DISEASE"** (*see* SCLERODERMIA).

**WHITLOW AND INFECTIONS OF THE HAND.**—A whitlow is an infection of a finger. Infections of the palm are very often secondary to whitlows, but may arise independently.

**Etiology.**—Infection of a finger in the majority of instances takes place from without, the organisms gaining entrance through a small wound, abrasion, needle prick, the tiny crack so often seen at the base of a tag of skin at the nail-fold, or through a sweat-gland. In the last case the disease usually appears on the palmar surface of the terminal phalanx. Occasionally a blood-stream infection occurs, but then as a rule the seat of origin is a bone (osteomyelitis).

Infections of the palm take place through wounds, or they are extensions from one of the fingers or thumb.

Infections of the dorsum are nearly always direct implantation infections.

**Varieties.**—The whitlow may be (1) subcuticular, (2) subcutaneous, (3) paronychial, (4) subungual, (5) thecal, or (6) osteal.

In the hand the infection is (1) thecal or (2) extrathecal.

**Symptomatology.**—The **subcuticular whitlow** is a minor affection; it is merely a purulent blister. Children are specially liable to suffer from it.

The **subcutaneous whitlow** is seen characteristically in the palmar surface of the terminal phalanx. It is a cellulitis beginning with

sharp pain and throbbing in the affected area which very soon becomes excessively tender, tense, and reddened. The pain is exceedingly acute, preventing sleep. The temperature may be raised. The severity of these symptoms is explained by the structure of the extremity of the finger. The skin is thick and connected with the periosteum by bands of fibrous tissue which limit the amount of swelling that can take place from a moderate inflammatory effusion. The exudate is therefore more or less confined under tension in a region which contains more sensory nerve-endings than any other part of the surface of the body. Because of these anatomical arrangements extension inwards to the bone is common and still more so because the nutrient arteries which go to the extra-epiphyseal portion of the terminal phalanx are liable to become thrombosed as they cross the inflamed area. Fluctuation is not a sign to which much importance is to be attached. The pulp of a normal finger fluctuates. The important clinical signs of suppuration are tenseness and extreme tenderness. Higher up the finger, opposite the second or first phalanges, a subcutaneous whitlow is liable secondarily to infect the subjacent tendon-sheaths.

The **paronychial whitlow** begins as a reddened painful, tender, swollen area at the fold of a nail. Pus forms, and often exudes from under the nail-fold. The condition is apt to be subacute or chronic. Extension is very likely to take place under the nail, thus giving rise to a **subungual whitlow**. This may also result from the prick of a needle, thorn, or splinter of wood passing under the free extremity of the nail, or from a primary infection of the underlying bone. Subungual whitlows are excruciatingly painful. The slightest pressure on the nail increases the pain. Sleep is impossible. Very soon the yellow pus is seen through the substance of the uplifted nail. Secondary extension to the bone is easy and not infrequent.

In the **thecal whitlow** the infection has spread from the subcutaneous tissue into the flexor synovial sheath. The sheaths of the index, middle, and ring fingers extend upwards from the terminal interphalangeal joints to the necks of the corresponding metacarpals. That of the thumb reaches upwards to a point about an inch above the upper border of the anterior annular ligament. In about 50 per cent. of subjects the synovial sheath of the little finger is likewise in communication with

## WHITLOW AND INFECTIONS OF THE HAND

the anterior common flexor sheath. Thecal suppuration is a very serious condition, which is likely to be followed, if not by destruction of the contained tendon, at least by adhesions limiting its movement. It is therefore essential to make a diagnosis and by immediate incisions give the patient the only chance of escaping these crippling after-effects. The *diagnosis* rests upon three signs: (1) exquisite tenderness limited to the anatomical situation and extent of the sheath, (2) more or less rigid flexion of the finger, (3) great pain on trying gently to extend the finger.

A suppurative process in a synovial sheath, more especially when the streptococcus is the invading organism, may extend through to the exterior of the sheath and particularly to the distal intermetacarpal intervals. Constitutional disturbance is pronounced.

When the infection is in the synovial sheath of the flexor longus pollicis, the tenderness is present above the anterior annular ligament on the radial side as well as below it in the palmar surface of the thumb. Should this synovial sheath communicate with the main anterior carpal sheath, the latter structure will be invaded by the inflammatory process. The anterior carpal flexor sheath can also be infected directly and by extension from a thecal whitlow of the little finger. The sac extends upwards to about  $1\frac{1}{2}$  in. above the anterior annular ligament, and downwards to the middle of the metacarpal bones. When infected, the wrist is fixed in flexion, a tender swelling is present both above and below the anterior annular ligament which marks a constriction at its middle, and any attempt to extend the fingers is accompanied by the severest pain.

When there is an **extrathecal infection** of the palm the process proceeds in one of the lumbrical spaces. If an abscess forms under the dense central palmar fascia, there is great pain and tenderness but no bulging in the

palm. The back of the hand becomes cedematous, and the pus eventually may reach the surface at the web between two of the fingers; or it may pass backwards through an interosseous space on to the dorsum of the hand. A very common place for an abscess to form

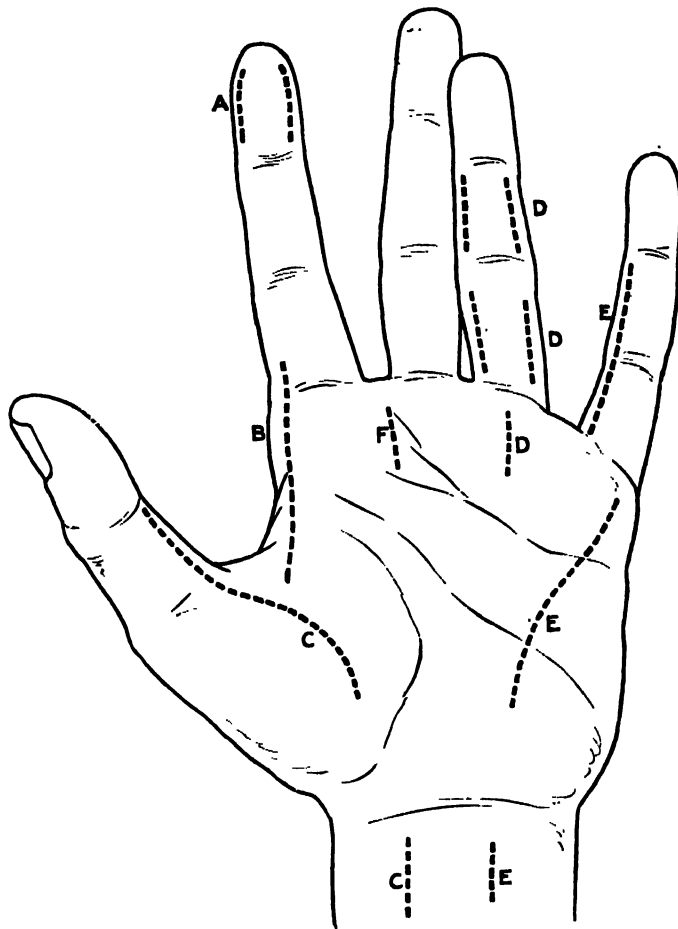


Fig. 106.—Operation for subcutaneous whitlow.

A, Incisions for subcutaneous whitlow of terminal phalanx. B, Incision for abscess of palm or thenar eminence. C, Incision for thecal whitlow of thumb. D, Incisions for thecal whitlow of index, middle, or ring finger. E, Incisions for thecal whitlow of little finger. F, Incision for abscess in a lumbrical space.

is in the thenar eminence between the thumb and index finger. This is because the lymph from the palm is collected into a single lymphatic vessel which traverses this area to reach the back of the hand before running up the arm.

Infections of the dorsum of the hand are not so common.

**Treatment.**—Although some infections of

## WHITLOW AND INFECTIONS OF THE HAND

the fingers subside under treatment and some surgeons recommend a wet alcohol dressing (under an impervious covering) or a wet, warm boric-acid dressing until there are actual signs of pus-formation, it is, on the whole, a better plan to institute active surgical measures too soon rather than too late. Necrosis of the terminal phalanx may take place very early owing to the anatomical arrangement of the tissues of the pulp referred to above; or extension to a tendon-sheath may occur in spite of careful observation. The rapidity of extension in streptococcal infections is sometimes surprising, yet this extension may be checked (though not invariably) by timely and well-placed incisions. Until this can be done, a hot boric-acid bath may be given or boric-acid fomentations applied. The affected part should be elevated to lessen congestion and pain. Carbolic acid should never be applied as a dressing to the fingers, from fear of gangrene supervening, and Bier's hyperæmic treatment should not be used until an incision has been made.

In **subcuticular whitlows** all that is necessary is to snip the purulent blister with scissors and to remove its outer wall. No anæsthetic is required.

In **subcutaneous whitlows** a local anæsthetic may be employed. A rubber constrictor is applied around the base of the finger and the four digital nerves or their neighbourhood are injected with 1-per-cent. novocain solution. The whole of the finger, including the bones, becomes anæsthetic in 10-15 minutes. This is a very convenient method, the only objection being that the tourniquet may cause a certain amount of pain until anæsthesia comes on. Unfortunately the injection is not invariably successful without the constriction unless adrenalin be added to the novocaine, yet this addition is proscribed because it is alleged that necrosis has followed a too prolonged adrenalin constriction of the digital arteries. If novocain be not used, nitrous oxide or ether should be employed. To open a whitlow after simply freezing the tissues with an ethyl chloride spray is as painful as with no anæsthetic at all.

Incisions should not be made directly in the middle line of the palmar surface of the fingers. If the pulp be infected, a lateral incision on the side of the greatest swelling, or incisions on each side, will drain the area; and when the wounds heal there will be no painful scar to interfere with the tactile functions of the finger. Incise freely laterally, but not, unless

absolutely necessary, opposite the creases in front of the joints (Fig. 106).

In **paronychia infections**, fomentations may be employed for a while, but, should pus continue to ooze out from under the nail, active measures should be taken. An incision with out removal of the nail is not satisfactory; it often leads to the spread of the pus under the nail. If the nail be removed early a new one of perfect shape will nearly always grow up but if this measure be neglected the resulting nail may be deformed and unsightly for life. Under general or local anæsthesia two small lateral incisions are made (Fig. 107), and the flap between them is pushed back, exposing the root of the nail. The nail should be gently prised up and removed. It is better to remove all the nail than merely the separated basal portion. Never incise the nail-bed. A small piece of gauze may be tucked between the flap and the nail-matrix for twenty-four hours.

When there is a **thecal sup-puration** the tendon-sheath must be drained forthwith. The operation should be done deliberately, under general anæsthesia, with the circulation arrested by a tourniquet around the upper arm. It should not be difficult by a careful clinical examination to be certain when the infection has reached the theca. But in case of doubt, rather than open a non-infected sheath, it is better to risk thrusting an exploratory hypodermic needle into it and to withdraw the contained fluid. Manifestly this puncture should be made at a point where the overlying tissues are least inflamed.

When the **sheath of the index, middle, or ring finger** is affected, lateral incisions should be made opposite the shafts of the first and second phalanges. It is better, if possible, to leave a bridge of the fibrous flexor sheath opposite the front of the first interphalangeal joint, to avoid prolapse of the contained tendons. The sheath, in the upper part of it, in front of the head of the metacarpal bone, should be opened in the middle line unless the disease has spread to a lumbrical space, when the opening should be made to this side (Fig. 107). The incisions in front of the phalanges may be bilateral.

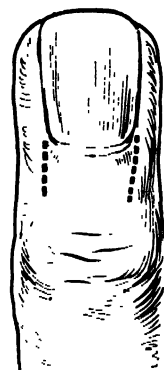


Fig. 107.  
Incisions for removal of nail.

## WHITLOW AND INFECTIONS OF THE HAND

When the **sheath of the thumb** is infected the anterior carpal sheath is almost certainly invaded as well. This is a very serious condition, which calls urgently for treatment. The operation (Fig. 108, A) is done under general anæsthesia. A tourniquet is applied to the upper arm and the operation carried out deliberately. The first skin incision is made along the middle of the flexor surface of the thumb opposite the first phalanx. It is then continued upwards over the thenar eminence towards a point a little to the inner side of the tuberosity of the scaphoid and within an inch of this protuberance. The lower and median margin of the flexor brevis pollicis is defined, and the interval between this muscle and the adductores obliquus and transversus opened up (for the relations of these structures see Fig. 108). In this interval lies the tendon of the flexor longus pollicis. Opposite the first phalanx the tendon may be exposed boldly, provision being made for leaving a bridge opposite the metacarpophalangeal joint. Above this bridge caution must be exercised, for two important structures here lie superficial to the tendon, viz. the superficial palmar arch and the cutaneous branch of the median nerve going to the lateral border of the thumb, whilst a little higher up still is the motor branch of the median going to the three outer thenar muscles. When a tourniquet is employed the structures can be seen and thus certainly avoided, the tendon being exposed as high as is consistent with their safety. Freer exposure can be obtained by incising the origin of the flexor brevis pollicis from the lower margin of the anterior annular ligament.

Attention has now to be turned to the carpal portion of the flexor longus pollicis sheath. An incision 2 in. long is made, extending upwards from the base of the styloid process along the

anterior border of the radius (Fig. 108, D). Care must be taken of the radial nerve, which winds round the radius and becomes cutaneous about 3 in. above the wrist-joint. The supinator longus tendon is defined, and this,

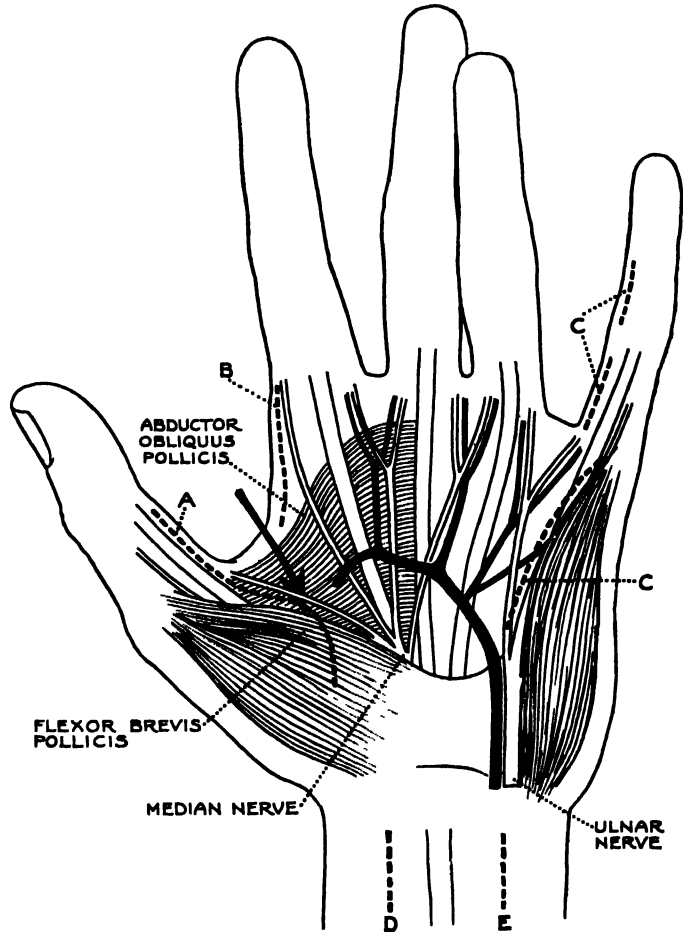


Fig. 108.—Incisions for exposing tendons of the hand, etc.

- A, Incision for exposing the flexor longus pollicis tendon. B, Incision for opening a thenar or middle palmar abscess. C, Incisions for exposing the tendon of the little finger. D, Incision above wrist for exposing flexor longus pollicis tendons from the front. E, Anterior incision for opening the main anterior carpal sheath. The arrow points to the interval between the abductor obliquus and flexor brevis pollicis, which must be opened up to expose the long flexor of the thumb.

together with the radial artery, is pulled forwards, exposing the flexor longus pollicis tendon and sheath. The sheath, if bulging, should be incised on its deep surface.

If thought advisable, an anterior incision may be made in addition. It begins just above the level of the tuberosity of the scaphoid

## WHITLOW AND INFECTIONS OF THE HAND

bone and runs upwards between the palmaris longus and the flexor carpi radialis. The flexor longus pollicis is then exposed from this aspect and its sheath opened. A hard drainage-tube must not be inserted into the lateral wound; it might press upon the radial artery and lead to secondary hæmorrhage. At most a narrow strip of rubber tissue should be inserted, and left for no longer than forty-eight hours. As a rule, no drain of any kind is necessary.

When the sheath of the little finger is infected, a long skin incision is made, under general anæsthesia, and after the application of a tourniquet, on the radial side of the finger from just above the distal crease to just below the metacarpo-phalangeal joint, and another from just above this joint on its ulnar side to a point one inch below and a little to the outer side of a vertical line passing through the pisiform bone (Fig. 108, c). The sheath is opened opposite the first and second phalanges, leaving bridges of the fibrous sheath opposite the first interphalangeal and metacarpo-phalangeal joints. Above the head of the metacarpal bone the outer margin of the flexor brevis minimi digiti is defined. If this muscle be retracted inwards the tendon-sheath will be exposed. The ulnar artery is retracted to the outer side before the sheath is incised, and the branch of the ulnar nerve going to the inner side of the little finger is avoided. The anterior carpal sheath must now be opened. An incision  $2\frac{1}{2}$  in. long (Fig. 108, e) is made along the anterior border of the ulna, beginning just above the lower extremity of this bone. The tendon of the flexor carpi ulnaris, the ulnar artery and nerve, and the long flexors are retracted forwards. The sheath is opened from the deep aspect. An anterior incision should also be made midway between the palmaris longus and flexor carpi ulnaris tendons. The same precautions about drainage should be taken as when the sheath of the flexor longus pollicis is opened (*see above*). Should this latter sheath, as well as the main flexor sheath, be infected, as it sometimes is, it also must be drained. A Spencer Wells forceps can be thrust from the ulnar incision under the flexor tendons and radial artery and cut down upon from the radial side. The flexor longus pollicis sheath can then be opened (*see above*).

The **osteal infection**, which usually begins in the terminal phalanx, calls for an incision down to the bone. Two lateral incisions, or a

U-shaped incision, passing over the extremity of the finger, are used. Should necrosis occur which is very likely, the dead portion of phalanx can be removed by turning down a flap outlined as above. The epiphysis at the base of the phalanx retains its vitality, and is left alone. After this operation the finger is useful but somewhat deformed at its extremity, the nail coming to lie farther forwards as a cap over the point of the finger.

**Palmar abscesses** are opened carefully under general anæsthesia and after the application of a tourniquet. The incision should lie over the most tender spot, usually in one of the lumbrical spaces. After the palmar fascia has been identified and divided, the closed points of a pair of Spencer Wells forceps are thrust into the abscess. A narrow strip of rubber tissue is used as a drain. A dorsal incision is called for only if the pus is pointing on this surface.

**Abscess of the thenar eminence** is opened by an incision along the radial border of the second metacarpal (Fig. 108, b). The centre of the palm can also be reached from this incision by passing inwards under the flexor tendons and lumbricals on the surface of the adductor transversus pollicis.

After incision of any of these abscesses intermittent wet dressings should always be used. Hot boric-acid fomentations are best. Intermittent immersion in boric-acid baths is also useful. In the case of the fingers it is a little awkward to avoid irritation of the surrounding skin if Dakin's solution be used, and in the case of the palm and anterior carpal sheath it is difficult to arrange Carrel's tubes satisfactorily.

As soon as the infection has been controlled, gentle active movements should be encouraged.

Stiff fingers following whitlows may be due to adhesions of tendons or ankylosis of the interphalangeal joints. Adhesions of tendons in their sheaths may sometimes be broken down under anæsthesia. It is not advisable to do this soon after a virulent streptococcal infection, from fear of lighting up the disease. A stiff extended finger with ankylosed joints is usually better removed, but before this is done an attempt at arthroplasty may be made. A useful movable joint cannot always be promised, but a sufficient number of successes has been obtained to justify the arthroplastic operation before removing the finger.

C. A. PANNETT.

## WHOOING-COUGH

**WHOOING-COUGH** (*syn.* Pertussis).—An acute specific contagious disease manifested by catarrh of the respiratory tract and paroxysms of coughing, followed by a long-drawn inspiration or "whoop."

**Etiology.**—The disease has a worldwide distribution, but is commoner and more severe in temperate climates than in warm or hot countries. It is mainly, though not entirely, a disease of early life. None of the commoner infectious diseases is so frequent in infancy, and especially during the first few months of life. It may even occur in the new-born, being transmitted to the fœtus in utero or acquired after birth from the mother suffering from the disease. Children of 1 to 5 years are most affected, and the morbidity is fairly high from 5 to 8 years, but after the age of 10, cases are exceptional. Adults not protected by a previous attack are as susceptible to infection as younger persons. The morbidity and mortality are both higher in girls than in boys. The disease is usually most prevalent and most fatal in March and April, and least so in September.

The infectivity is highest in the catarrhal stage, and rapidly declines when the cough has become paroxysmal. Transmission of infection is almost always by direct contact. The vitality of the specific micro-organism outside the body is shortlived, and therefore fomites such as clothes, toys, and books play little part in its dissemination. It is more probable that carriers may be responsible to some extent for the spread of the disease (*see* CARRIERS OF INFECTION).

**Morbid anatomy.**—There are no characteristic naked-eye lesions. Hyperæmia of the mucous membrane of the larynx, trachea, and bronchi is constant. In fatal cases bronchopneumonia and emphysema are almost invariable, and tuberculosis of the bronchial glands is common. There is usually some dilatation of the right side of the heart. Capillary hæmorrhages and œdema may be found in the brain and meninges.

Mallory and Horner have claimed that the specific lesion in pertussis consists in the presence of a large number of bacilli between the cilia of the epithelial cells of the trachea and bronchi. The action of these bacilli, which resemble those described by Bordet and Gengou, would thus appear to be mainly mechanical, in that their presence interferes with the action of the cilia and checks the normal process of secretion, as well as being

a constant source of irritation to the laryngeal and bronchial mucosa.

**Bacteriology.**—The micro-organism generally accepted as the causal agent was first described by Bordet and Gengou in 1906. The *Bacillus pertussis*, an ovoid, non-motile, Gram-negative cocco-bacillus, is found in great numbers in the sputum in the early stage, but becomes more scanty as the disease progresses. It is not found in the blood, except just before death. It grows chiefly in the lung, and rarely above the larynx. Owing to its slow growth it is easily overgrown by pneumococci and influenza bacilli. The best culture-medium is defibrinated blood mixed with an equal quantity of 3-per-cent. agar containing a little extract of potato or glycerin.

The serum of the pertussis patient agglutinates the bacilli. Bordet and Gengou obtained a complement-fixation test by using an emulsion of the organism and the serum of the patient's blood.

By injection of cultures of the Bordet-Gengou bacillus, Klimenko in 1908 produced an infection of the respiratory tract in monkeys, with symptoms more or less closely resembling pertussis but without the peculiar whoop, and obtained the organism again from their trachea and lungs. He was also able to demonstrate the complement-fixation test with their blood. I. Inaba (1913) also successfully inoculated a monkey by swabbing the naso-pharynx with Bordet-Gengou cultures, and recovered the bacillus in film preparations and cultures from the sputum.

**The blood.**—The characteristic blood-change in pertussis is a leucocytosis with a predominance of the lymphocytes. Cases with a low leucocyte count show a relatively high lymphocytosis and a marked decrease of polymorphonuclears. There is less obvious increase in the mononuclears and transitionals.

This blood-change appears early in the catarrhal stage, and therefore possesses considerable diagnostic value.

Slight eosinophilia occurs in convalescence.

**Symptomatology.**—Owing to the indefinite nature of the onset, the exact duration of the incubation period is difficult to determine, but it is generally regarded as not exceeding a fortnight, and sometimes as being as short as three or four days.

Although it is not always possible to differentiate them in practice, it is usual to distinguish three periods in the description of the disease.

## WHOOPING-COUGH

1. **Catarrhal stage.**—Apart from the examination of the blood, the onset of whooping-cough presents nothing characteristic. The patient appears to be suffering from a severe cold. There is frequent sneezing, the voice is hoarse, and the eyes are suffused. The cough is hard and dry, most frequent at night, and is little affected by ordinary cough-mixtures. There is usually little if any constitutional disturbance. Either the temperature is not raised at all, or there is a slight rise at night with a fall to normal in the morning.

2. **Paroxysmal stage.**—After a period varying from a few days to a fortnight the cough gradually becomes paroxysmal in character. The typical paroxysm consists of a series of noisy and violent expirations following each other in rapid succession, and ending with a long-drawn crowing inspiration, the "whoop." The paroxysms continue until the expulsion of a more or less abundant viscid and stringy mucus, frequently accompanied by vomiting.

During the paroxysm the patient clings for support to the nearest object, the face is swollen and cyanosed, the tongue protruded, the eyes prominent, and the veins of the neck turgescient. The pulse rises to 120 or 150. In young children the urine and feces are frequently voided. The friction of the tongue against the lower incisor teeth gives rise to a sublingual ulcer which, though not absolutely pathognomonic, is more frequently found in pertussis than in any other disease.

The average number of paroxysms is from fifteen to twenty in the twenty-four hours, but they may never exceed three or four daily throughout the attack or, on the other hand, may take place every half-hour. They may occur spontaneously or be provoked by some irritation or emotion. Older children receive warning of the oncoming of an attack by a tickling sensation in the throat, retrosternal pain, nausea, or giddiness. Except after severe attacks, which are followed by exhaustion, the child quickly recovers his normal condition after expectoration of the mucus, but a certain turgescence of the face tends to persist between the attacks and thus affords a valuable help in diagnosis. Attacks of sneezing may sometimes take the place of a typical paroxysm. In young infants and sometimes in adults the whoop may be absent throughout. The urine during this stage has a high density owing to an excess of uric acid. Albuminuria is exceptional, but sugar is sometimes present.

3. **Period of decline.**—After a period usually

of about six weeks' duration, but often considerably longer in neuropathic subjects, the paroxysms diminish in number and severity; vomiting ceases, and the whoop is no longer heard. Finally, the cough loses its paroxysmal character, and the sputum becomes loose and muco-purulent.

Occasionally a transient recrudescence of paroxysms may occur at this period, especially if the patient develops bronchitis. A true second attack of whooping is extremely rare.

**Complications.** 1. **Respiratory.**—The most important complication is *broncho-pneumonia* which is more frequently associated with whooping-cough than with any other acute infection except measles. It is most frequent during the first two years of life, and is mainly responsible for the high mortality of pertussis at this age. It usually occurs during the paroxysmal stage, rarely during the period of invasion or of decline. Its occurrence is generally followed by a diminution in the frequency and intensity of the paroxysms and the suppression of the whoop. Death may occur from asphyxia or convulsions in four or five days. On the other hand, the broncho-pneumonia instead of resolving, may become chronic, and in such cases bronchiectasis is liable to result.

*Lobar pneumonia* is a much less frequent complication. Pleurisy and empyema are also rare.

*Vesicular emphysema* is not uncommon, being due to the violent distension of the pulmonary vesicles while the glottis is spasmodically narrowed during the expiratory paroxysms. *Interstitial emphysema*, resulting from the rupture of the distended vesicles, is much rarer. It may extend to the mediastinum, the cellular tissue of the neck and thorax, and even give rise to general subcutaneous emphysema.

2. **Nervous complications.**—*Convulsions*, which are the most important nervous complication of whooping-cough, are practically confined to children in the first three years of life. They may occur in association with the paroxysms or independently. They are usually met with in cases already complicated by broncho-pneumonia, but are occasionally found when no pulmonary disease is present. They may be localized to the face or a single limb, or be general. The cerebro-spinal fluid obtained by lumbar puncture—which has sometimes proved of therapeutic value—is under hypertension, but contains no albumin and few cells. If the convulsions be repeated, death almost invariably occurs, but a single attack is often followed by recovery.

## WHOOPIING-COUGH

*Spasm of the glottis*, the *convulsions internes* of the French, usually takes place during a paroxysm, much more rarely between the attacks.

*Cerebral paralyses* are much rarer than convulsions, and occur in older patients. They consist of hemiplegia, aphasia, and palsies of the cranial nerves. Usually no gross lesions are found at autopsy, but only hyperæmia, serous infiltration of the meninges, or fine hæmorrhages in the brain substance such as occur in other infectious diseases. Such findings indicate a toxic rather than a mechanical origin for the symptoms.

Other nervous complications are still more uncommon, but a few cases of peripheral neuritis, Landry's paralysis, Friedreich's disease, and disseminated sclerosis have been reported after whooping-cough.

**3. Cardiac complications.**—*Dilatation and hypertrophy* of the heart, especially affecting the right side, are frequent. Permanent damage may result.

**4. Alimentary system.**—*Catarrhal stomatitis* is not uncommon. *Ulcerative stomatitis*, apart from the characteristic sublingual ulcer, is less frequent. Vomiting is usually associated with the paroxysms, but may occur independently as an indication of *catarrhal gastritis*, which may be caused or aggravated by swallowing the sputum. *Profuse diarrhœa*, the indication of enteritis, may also occur.

**5. Hæmorrhages.** Various hæmorrhages may occur during the paroxysms; of these the commonest are epistaxis, bleeding from the gums, and petechiæ in the skin. Subconjunctival hæmorrhage is met with in about 2 per cent. of the cases (Ker), and is sometimes accompanied by palpebral ecchymosis. Rarer varieties are hæmorrhages from the ear due to rupture of the tympanum, hæmoptysis, hæmatemesis, and meningeal or cerebral hæmorrhage.

**Association with other diseases.**—Whooping-cough may be associated with any other infectious disease, but has a special predilection for *measles*. Epidemics of whooping-cough and measles often occur simultaneously. Subjects of pertussis are very liable to contract measles, and vice versa.

In cases of concurrent pertussis and *diphtheria* the incidence of croup is higher than usual. In tracheotomy cases the onset of violent paroxysms after removal of the tube may necessitate its immediate replacement and render its stay in the trachea unusually protracted. The occurrence of any febrile

disease is usually, though not invariably, followed by a diminution in the number of the paroxysms and suppression of the whoop.

**Diagnosis.**—In the catarrhal stage a certain diagnosis of whooping-cough is impossible, but the persistence of a troublesome cough unaffected by treatment and worst at night, with little or no physical signs to account for it, is extremely suggestive, especially after exposure to infection. Further help in diagnosis is given by examination of the blood, which at this stage shows a leucocytosis with marked predominance of the lymphocytes.

The development of paroxysms ending with a whoop and the expectoration of viscid mucus leaves no doubt as to the nature of the case. In the intervals between the paroxysms the presence of a sublingual ulcer and a bloated and congested face are corroborative evidence.

Diagnosis of whooping-cough by discovering the Bordet-Gengou bacillus in the sputum or by the complement-deviation test requires an expert bacteriologist.

The conditions most likely to be mistaken for whooping-cough are enlargement of the bronchial glands, acute bronchitis and broncho-pneumonia. The paroxysmal cough due to pressure of *enlarged bronchial glands* on the vagus is distinguished from pertussis by the prominence of the superficial veins on one or both sides of the neck, impairment of resonance at the second or third intercostal space near the sternum with harsh tubular breathing, and the presence of Eustace Smith's murmur—a to-and-fro murmur heard on one or other side of the manubrium when the head is thrown back. Further, the glandular enlargement being usually tuberculous, other signs of tuberculosis may be present, especially an irregular pyrexia, which is not found in uncomplicated whooping-cough. Lastly, the characteristic whoop does not occur.

A paroxysmal cough may also be met with in *acute bronchitis* and *broncho-pneumonia*, but is of brief duration and is not accompanied by vomiting or a "whoop."

**Prognosis.** The prognosis mainly depends upon the age of the patient and the occurrence of complications, especially broncho-pneumonia and convulsions. Uncomplicated cases usually recover.

In the winter, death is most frequently due to broncho-pneumonia, whilst in the summer, enteritis may also be responsible for some of the deaths.

Rickety children are specially bad subjects



## WHOOPING-COUGH

owing to their liability to broncho-pneumonia. The irritation caused by the presence of adenoids also aggravates the disease and favours its prolongation.

The remote prognosis must be guarded, owing to the liability of the whooping-cough patient to develop tuberculosis or to have pre-existent and dormant tuberculosis awakened into fresh activity.

**Prophylaxis.**—The patient should be isolated until the termination of the paroxysmal stage or, at least, for as long as expectoration continues. The disease being most contagious in the period of invasion, children with catarrhal symptoms in a household or hospital ward in which a case has recently occurred should be regarded with suspicion and kept apart from those who have not had the disease.

It is most important to protect from infection children under 2 years, because of the high mortality and frequency of complications during this period; it is in such cases especially that the prophylactic use of pertussis vaccine may be of service (*see below*).

The sputum and vomit should be disinfected as carefully as in pulmonary tuberculosis, but disinfection of the room is unnecessary owing to the low vitality of the Bordet-Gengou bacillus.

**Treatment.** (a) **General.**—All whooping-cough patients require plenty of fresh air. Unless the temperature is raised or the paroxysms are severe and followed by exhaustion, the patient need not remain in bed. The bowels must be kept open. All causes of excitement should be avoided, and sources of local irritation, especially adenoids, removed. A readily assimilable diet should be ordered. Dry, crumbly substances likely to cause pharyngeal irritation, as well as indigestible food, should be forbidden. When vomiting occurs directly after a meal as the result of a paroxysm, the food should be repeated in about ten minutes. Nutrient enemata are rarely required. During the paroxysm the child should be helped to rid itself of the viscid mucus. In severe cases Naegle's method of averting the attacks by pulling the jaw downwards and forwards is to be recommended. Change of air, especially to a sea climate, is advisable in convalescence.

(b) **Drug treatment.**—Many cases require no drug treatment whatever. In pertussis, as in diphtheria during the pre-antitoxin era, the multiplicity of remedies is evidence of their inefficacy. All that they can effect is an

alleviation of the symptoms. Expectorants and sedatives are chiefly employed. Of the former the most useful is vinum ipecacuanhae given in 5-min. doses every four hours; of the latter, tincture of belladonna, starting with doses of 1 to 2 min., gradually increased until the pupils are dilated; antipyrin in doses of 1 gr. for each year of age; sodium bromide 5 gr. t.d.s.; benzyl benzoate 5–40 min. of 20-per-cent. solution t.d.s.; and a combination of veronal  $\frac{1}{2}$  gr. and quinine sulphate 1 g t.d.s. Some relief may be obtained by inhalations of carbolic acid, cresolene, or eucalypti oil. Intramuscular injections of ether (1 c.c. up to the age of 8 months and 2 c.c. for older children) every two days have recently been highly recommended by French authorities.

(c) **Vaccine treatment.**—Within the last few years a pertussis vaccine has been used both as a prophylactic and as a curative agent. The vaccines employed are prepared from cultures of the Bordet-Gengou bacillus, either alone or in combination with cultures of the influenza bacillus, hæmophilic bacilli, and streptococci. The doses range from 20 million to 50 million or 100 million bacteria. The injections, which are given every three or four days, produce little or no local reaction and only a slight rise of temperature. The results hitherto obtained, though encouraging, are not conclusive. The prophylactic value of pertussis vaccine is by no means so high as that of typhoid vaccine, as is shown by the fact that out of 244 children to whom Hess gave protective doses, 20 developed whooping-cough. In estimating the value of the curative power of vaccine, the number of naturally abortive cases must be taken into account, as well as the suggestive influence exercised by the injections themselves. No bad results, however, have followed their use, and, apart from the expense, there appears to be no objection to a wider trial being given to this method of treatment.

J. D. ROLLESTON.

**WIDAL'S REACTION** (*see* SEROLOGICAL DIAGNOSIS).

**WINCKEL'S DISEASE** (*see* Epidemic Jaundice of Infants, under JAUNDICE; URINE, EXAMINATION OF).

**WISDOM TOOTH, IMPACTION OF** (*see* ORAL SEPSIS).

**WOOL-SORTER'S DISEASE** (*see* ANTHRAX).

## WOUNDS, TREATMENT OF

**WORMS** (*see* **INTESTINAL WORMS**).

**WOUNDS FROM THE MEDICO-LEGAL STANDPOINT** (*see* **INJURIES FROM THE MEDICO-LEGAL STANDPOINT**).

**WOUNDS, TREATMENT OF.**—The wounds considered here are the open wounds met with in civil practice and certain punctured wounds, but wounds of the abdominal wall and viscera receive attention in the article on **ABDOMINAL INJURIES**, and those of the scalp in **SCALP WOUNDS, TREATMENT OF**.

Wounds are described as incised, lacerated and contused, and punctured. Lacerated and contused wounds are the most serious, because the vitality of the cells of the margin, from which repair begins, is depressed. Punctured wounds may give trouble because, perhaps, the infection is implanted deep from the surface. Injury to a large blood-vessel may be a serious complication; and nerves, bones, and tendons may also be damaged.

All accidental wounds are necessarily infected, and this fact militates against healing. The **essentials for proper repair** are—

1. The arrest of hæmorrhage.
2. Rest to the injured part.
3. Coaptation of the sides of the wound, with obliteration of all dead spaces.
4. Diminution in the dose of the implanted infection.

Of these, the most difficult to secure is the last, and the several methods of wound treatment are all endeavours to combat infection by different means. Complete bacteriological sterilization is seldom if ever attained. Fortunately the tissues are able to deal with infection if serous fluid is not allowed to collect. In such exudate certain bacteria, in particular staphylococci and streptococci (serophytes), flourish, and get a firm footing before leucocytes can assemble in sufficient numbers to destroy them. Hence the rule that no dead spaces must be left, or, if cavities are unavoidable, stagnation of the discharges in them must be prevented by the provision of efficient drainage. For the same reason it is equally important to check oozing so far as is possible. For ligating vessels in an accidental wound, catgut must be used, never silk, and non-chromicized catgut is best. Suturing may be performed within a few hours of the accident (primary suture), within two or three days (delayed primary suture), or later (secondary suture).

Certain general factors of success in treat-

ment must be borne in mind. The healing powers of the patient may be lowered by loss of blood, an acute infection, or some chronic disease, especially chronic interstitial nephritis and diabetes. Loss of blood may call for transfusion (q.v.), and the general disease must receive attention in every case. When a wound is contaminated with ground dirt, anti-tetanic serum should be administered (*see* under **TETANUS**). Splints may be necessary to keep the injured part at rest.

**Primary excision of the wound, with immediate suture.**—This is followed by a large percentage of successes if it is carried out in properly selected cases. The wound should temporarily be plugged, and the surrounding skin cleansed and disinfected. Soap followed by alcohol and by ether is used for this purpose. The whole of the wound is then excised, so that none of the original wound surface remains. The instruments used are now discarded for a fresh set. After hæmostasis the wound is sutured. It is clear that this procedure can only be carried out when important structures do not lie exposed in and form part of the wall of the wound—that is, nerves and large vessels must be respected. When from this cause the ideal procedure cannot be carried out, it is sometimes possible to do *delayed primary suture*, but this can be done only when steps have been taken to destroy the remaining infection (*see* below). Delayed primary suture is also appropriately done when it is known that the primary infection has been great, even though the whole wound has been excised.

**B.I.P.P. treatment.**—This procedure, devised by Rutherford Morison, is useful in treating contaminated wounds. The surrounding skin is scrupulously cleansed and all obviously contaminated tissue removed by means of the knife. Hæmostasis is carefully effected, the wound dried with pieces of gauze, bathed with alcohol, and smeared over with bismuth and iodoform paste. It is then sutured without drainage. The formula of B.I.P.P. is given as bismuth subnitrate 1 part, iodoform 2 parts, liquid petrolatum q.s. to make a thick paste. This paste need not be specially sterilized. If all goes well the dressings are not removed for a week. A brown-coloured discharge in the dressing is to be expected. A cleansed wound which it is thought not desirable to close at once may be smeared over with the paste and left open to be sutured after a day or two.

The **Wright treatment** of wounds by hyper-

## WOUNDS, TREATMENT OF

**tonic saline** is useful when the amount of infection has been great, and also in wounds at a later stage when sloughing has occurred. In this treatment it is recognized that there are two classes of infecting microbes—(1) a large group, including the gas gangrene microbes, which cannot grow in unaltered serum or blood, but only in wound discharges that have lost their antitryptic power or their normal degree of alkalinity (sacro-saprophytes); and (2) a small group, composed of staphylococci, streptococci, and a diphtheroid bacillus, which flourish in unaltered serum or blood (serophytes). The former group is killed by unaltered serum, the latter group by the phagocytosis of leucocytes. The object of the treatment is to establish a flow of unaltered serum and to encourage leucocytic emigration. It is done in two stages. Hæmostasis having been effected and all grossly contaminated tissues removed, the wound is lightly packed with gauze soaked in 5-per-cent. sodium chloride solution, and over the whole is put an impervious covering to keep the dressing moist. This dressing should be changed every four hours. In some patients irritation of the surrounding skin is caused by the saline solution; in such persons the neighbouring skin should be smeared over with sterile vaselin. Under this treatment sloughs are rapidly removed and a flow of fresh serum is promoted. When all dead tissue has disappeared and the wound surface is covered all over with granulations (three or four days, or more), it is useless to continue with the hypertonic solution, which now will do harm rather than good because it inhibits leucocytic emigration and tends to disintegrate white cells that have left the tissues. The formation of pus must now be allowed to proceed by substituting normal saline for the hypertonic solution. But always it is essential that there should be no stagnation of the tissue fluids in the wound. Drainage must be free and effective. Pocketing in the wound is fatal to physiological sterilization.

Wright has shown that it is not necessary to wait for the disappearance of streptococci or for complete sterilization of a wound before attempting suture. On the contrary, by proper apposition of the walls of the wound the tissues are placed in more advantageous circumstances for combating the infection. Suture may be attempted when, all sloughs having disappeared entirely for some days, the whole of the wound surface is covered by granulations, and when there has been for a

like period a properly established effusion of the "laudable" pus of the older surgeons—provided always that such exudate has been allowed to drain away, for the stagnation of the wound discharges creates circumstances eminently favourable to bacterial growth. In suturing, all dead spaces must be obliterated. To accomplish this, deep catgut sutures may be necessary. If, however, the wound edges can be brought into apposition by strips of strapping placed transversely across them, this expedient is preferable to sutures. The epithelial margin of the wound should be excised with the knife before doing it. When a wound has been very septic, passing a suture through its sides may light up a waning infection. In such wounds thick silkworm-gut mass sutures may be inserted with a large curved needle so that the whole wound, including its deepest part, is encircled; these sutures are left untied until the lapse of three or four days has demonstrated that no aggravation of the infection is to be feared.

**The Carrel-Dakin treatment.**—An outline only of this treatment can be given here. The method is an extremely valuable one, but requires a great deal of care and attention. A nurse skilled in all the details of the method is necessary, and the solution used must be made up with a scrupulous observance of the originators' instructions; otherwise failure to attain results is inevitable, and it is better to follow Wright's technique, which gives results equally good, except in a few cases in which the conditions are not favourable, e.g. empyema. In the Carrel-Dakin treatment eradication of the infection is accomplished by two-hourly irrigation of the wound with a solution of sodium hypochlorite. This solution must not be above 0.5 per cent. or below 0.45 per cent., and there must be no free caustic soda present. It is made up as follows:—

1. Place in a 12-litre flask 184 grm. of chloride of lime and 5 litres of tap-water, shake vigorously, and leave all night.
2. Make a solution of 92 grm. of anhydrous carbonate of soda and 76 grm. of bicarbonate of soda in 5 litres of cold water.
3. Add the soda solution to the chloride-of-lime solution, shake, and allow to settle.
4. After half an hour, siphon off and filter the clear fluid. It should be kept in the cold and away from light.

## WOUNDS, TREATMENT OF

Unfortunately, the composition of chloride of lime is variable, so the above quantities will only be correct when the percentage of available chlorine is 25. As it is essential to use the quantitatively correct solution the percentage of available chlorine must be estimated in every batch of chloride of lime used. This is a recognized chemical procedure, the method of performing which can be learned by referring to any book on analytical chemistry.

The percentage of available chlorine being known, the necessary quantities are obtained by referring to the following table :—

Titration of chloride of lime Cl per cent.	Quantities to be used to obtain 10 litres of solution of hypochlorite of 0.475 per cent.		
	Chloride of lime	Anhydrous sodium carbonate	Sodium bicarbonate
20	230	115	96
21	220	110	92
22	210	105	88
23	200	100	84
24	192	96	80
25	184	92	76
26	177	89	72
27	170	85	70
28	164	82	68
29	159	80	66
30	154	77	64
31	148	74	62
32	144	72	60
33	140	70	59
34	135	68	57
35	132	66	55

The solution is placed in a container hung at the side of the patient's bed, 3-4 ft. above the level of the wound.

A single rubber tube provided with a clamp conveys the solution to a glass connexion with two or more side branches, to which are attached the irrigating tubes proper. These are red rubber tubes with an internal diameter of 4 mm. perforated with a number of holes  $\frac{1}{2}$  mm. in diameter. The ends of the tubes are closed by a ligature. The wound receives the usual preliminary cleansing as in Wright's method, but dependent drainage is avoided as the method can only be used effectively when the wounds can be filled by the solution. As many tubes must be used as are necessary to conduct the fluid to every recess in the wound. This is essential. The tubes must be in contact with the surface of the wound, and

to keep them in contact the centre of the wound cavity between the tubes is lightly packed with gauze; but none of this gauze must intervene between tubes and wound wall. If the fluid is to pass equally abundantly along each tube, the holes in the different tubes attached to the same glass connexion must be equal in number. The irrigation tubes being in place, the glass connexion is strapped to the neighbouring skin. This skin round the wound is liable to be irritated by the solution, so it is protected by strips of gauze soaked in melted sterilized vaselin. Over the whole is placed a wool dressing. Every two hours day and night the nurse releases for a few seconds the clamp on the tube attached to the reservoir, allowing an instillation of 10 or 15 c.c. to take place. The wound should not be flooded. Every day the gauze in the wound and the external dressing are changed, and films are made from the worst-looking parts of the wound. By examining these the progress of sterilization can be watched.

The wound is ready for suture when not more than one microbe in four or five microscopic fields can be seen.

From the above account it will be realized that the Carrel-Dakin treatment requires infinite care and attention to details; but the technique is not difficult to learn, and when carried out gives excellent results. Instead of the hypochlorite solution, chloramine-T may be used in 0.2-per-cent. solution. The reports with this antiseptic are not so good as with the original hypochlorite solution. Eusol should not be substituted.

**Bier's treatment.**—This method is still used somewhat extensively abroad, but has been discarded by most English surgeons. The process is described in a separate article. Hyperæmia is also induced by application of the ordinary boric-acid fomentations.

**Antiseptics.**—Whilst it is an undoubted truth that antiseptics are of immense use in surgical technique outside the wound and on the unbroken skin, the most reliable work tends to show that the indiscriminate flushing out of wounds with solutions of antiseptics does harm rather than good. The Carrel-Dakin and the B.I.P.P. treatment are exceptions to the general rule that antiseptics are injurious when brought into contact with the wound surface. Certain of the newer antiseptics, however, must be mentioned.

**Eusol.**—A mixture of equal parts of dry bleaching powder and boric acid is made;

## WOUNDS, TREATMENT OF

this powder is called *eupad*. It must be kept in a tightly stoppered bottle. To make a solution of eupad, 25 grm. of eupad are added to one litre of water. The solution is filtered after shaking and allowing to stand. This solution of hypochlorite is too irritating for use in wounds, though it has been extensively employed and highly extolled.

*Acriflavine* and *crystal violet* are antiseptics having a selective action upon staphylococci and streptococci. Both may be used in a strength of 1:1,000 for irrigating wounds and soaking the gauze which fills them. Acriflavine is useful in the early stages. If its application is continued it tends to delay repair. Acriflavine has another drawback, as it interferes very materially with phagocytosis. Unlike most other antiseptics, it acts more powerfully in serum than in aqueous solution. *Brilliant-green* (1:1,000) is another powerful antiseptic; it has been much extolled in the treatment of wounds.

**Treatment of granulating surface wounds.**—Where a large area of skin has been destroyed and is lacking, the final stages of repair and complete epithelialization of the wound are very slow, and may be delayed indefinitely. In civil practice burns frequently enter this stage and remain but little altered for long periods. It is in such cases that skin-grafting is called for. Before resorting to it, however, natural healing must be assisted in every manner possible.

The dressing of these granulating wounds is important. If dry gauze is used it adheres to the surface, and its removal not only causes very severe pain but damages the underlying granulations, which bleed, and injures the advancing epithelial margin. When such dressings are stuck, remove all but the layer next the granulations, and cover this thickly with sterilized vaselin. In twenty-four hours it will be possible to remove the gauze without pain. When the wound surface is clean, boric ointment ( $\frac{1}{4}$  B.P. strength) or eucalyptus ointment (1 dr. to 1 oz. of yellow paraffin) makes a useful dressing. Another method is to cut a piece of thin perforated celluloid larger than the wound by about  $\frac{1}{2}$  in. all round; this is kept in place by strips of zinc-oxide plaster; over the celluloid is placed a compress of gauze soaked in normal saline, and a sheet of oiled silk. The wound can then be irrigated with normal saline twice daily. This is an excellent mode of treatment.

Sometimes granulations become exuberant

and grow up above the level of the advancing epithelium; they then form a barrier across which the young epithelium is not able to advance. The zinc oxide adhesive method of treatment is useful in these circumstances. Strips of zinc-oxide plaster  $\frac{3}{4}$ –1 in. wide are arranged along the edges of the wound, one half the width being on the skin and the other half lying upon the granulations. The rest of the wound is covered with an ointment spread upon gauze. The strapping is left on for twenty-four hours. It is not applied continuously, but on alternate days only, the wound being dressed with a simple ointment on the intervening days.

*Scarlet-red* is an epithelial stimulant and is sometimes used with advantage. An ointment in yellow paraffin containing 8 per cent. of the dye is spread upon a piece of lint so cut that when applied to the wound it just overlaps the growing epithelial edge by about  $\frac{1}{4}$  in. all round. The surrounding skin is smeared over with weak boric ointment ( $\frac{1}{4}$  B.P. strength). As scarlet-red is a little irritating, it can only be used for six days in succession; on the seventh day the boric ointment dressing only is applied.

Exuberant granulations may be cut off with scissors curved on the flat, or reduced by the application of a solid silver-nitrate stick.

**Skin-grafting.**—There are three methods employed:

1. Reverdin's method of small deep grafts.
2. Thiersch's method of larger superficial grafts.
3. Whole-skin thickness grafts.

1. *Reverdin's method* gives perhaps the highest percentage of successful results. The area to be grafted, if it is not a fresh operation wound, must be rendered as aseptic as possible. The Carrel-Dakin irrigation employed for some days, until the bacterial count is reduced to one in four or five fields, will effect this, but good results may be got without preliminary irrigation in healthy granulating wounds. If the Carrel-Dakin treatment is used, it is continued up to the time of operation. When some other dressing has been applied, gauze soaked in normal saline should be substituted for twenty-four hours before the grafting takes place, and this wet dressing should be changed every four hours, the wound secretions being washed away at each dressing. It is better to lay the grafts upon the unaltered granulations; they adhere quite well, whilst if the

## WOUNDS, TREATMENT OF

granulations are scraped away it is difficult to get complete hæmostasis. One of the factors of success is that the bed should be dry and that no blood should collect under the freshly applied grafts.

The first step in the operation is to prepare the bed. The granulating area is irrigated carefully with normal saline, mopped gently to remove excess, and covered over with dry gauze. It is then left while the grafts are being cut. The outer, inner, and anterior aspects of the thigh are the most convenient situations from which to remove the grafts. Antiseptics should not be used in the preparation of the skin; soap-and-water followed by alcohol is sufficient. A tiny cone of skin is picked up on the point of a needle and the base of the cone cut across with a sharp scalpel.

The graft should be at most  $\frac{1}{4}$  in. in diameter; in its central part it is composed of the whole thickness of the skin. Adhering to the point of the needle, it is transferred to the area to be grafted, the dry gauze having been removed, and it is pressed down gently upon the granulations. The grafts should be placed closely together, an interval of about  $\frac{1}{8}$  in. remaining between them. Great care should be taken not to convey sepsis from the wound to the area from which the grafts have been taken. Several needles held in Spencer Wells forceps should be employed so that they can be re-sterilized before each transference of a graft is made.

The operation may be done under local conduction anæsthesia. A 2-per-cent. solution of novocain is required, to which is added adrenalin solution (1 : 1,000) in the proportion of one drop for every drachm. It is necessary to block two nerves, the external cutaneous and the anterior crural. When only a small area is to be grafted it may be sufficient to anæsthetize the external cutaneous nerve only. After this an area on the outer side of the thigh about  $2\frac{1}{2}$  by 2 in. becomes insensitive. After both nerves have been blocked the whole of the inner, anterior, and outer aspects of the thigh are available for cutting grafts. The external cutaneous nerve is blocked by infiltrating transversely about 1 in. below the anterior superior spine of the ilium both in the superficial fascia and just beneath the deep fascia. Five c.c. of the solution are used. The anterior crural nerve is injected just below Poupart's ligament about  $\frac{1}{2}$  in. externally to the pulsating femoral artery. Five c.c. of

the novocain solution are again used. The nerve is beneath the deep fascia. If pain is felt radiating down the thigh, perhaps to the inner side of the ankle, the operator knows that the point of the needle is in the nerve.

The best dressing after skin-grafting is made as follows: A piece of thin perforated celluloid is cut of sufficient size to overlap the wound well all around. It is placed over the grafts and maintained in position by strips of strapping along its edges. Over this is put gauze wet with normal saline, and on top of all a piece of impervious protective. Twice daily the gauze should be replaced and the wound irrigated with saline without removing the celluloid sheeting, which is not taken off for eight days, when a simple ointment dressing will probably suffice.

It is said that mosquito netting dipped in molten paraffin makes a good substitute for the perforated celluloid sheeting. If it be sterilized in the autoclave with the other dressings the solidified paraffin which stops up the meshes will disappear, but enough wax will remain in the fabric to prevent it from sticking and absorbing wound secretions.

2. *Thiersch superficial grafts.*—The area to be grafted should be prepared as described above. The grafts are similarly obtained from the thigh.

Thiersch grafts are cut much larger than Reverdin grafts, either with the special large knife or an ordinary sharp razor moistened with saline. When they are properly cut, the surface left will present a number of tiny bleeding-points, the apices of severed papillæ of the corium. The whole of the area to be grafted should be covered by these thin grafts, which overlap the margins of the wound. If it is larger than  $\frac{1}{2}$  in. square, V-shaped slits should be cut in the margins to prevent accumulation of secretion under the grafts, which is the cause of many of them not "taking." The wound is best dressed with the perforated celluloid as described above.

The resulting scars after Reverdin grafting are stronger and more resistant to injury than those after Thiersch grafting, but the cosmetic results are not so good. The Reverdin grafted area has the appearance of many slightly elevated pale areas (the grafts) with a reddened depressed scar network between them.

3. *Whole-skin thickness grafts.*—It is not so easy to get a successful result by this method, but when obtained the grafted area is far superior in appearance and trauma-resisting

## XANTHOMA

properties to that obtained after the other procedures. The skin should be taken from the inner side of the thigh or the inner side of the upper arm if skin devoid of hairs be required. Generally the graft should be spindle-shaped and not too large to prevent suturing of the resulting wound. The whole skin down to the fat is removed. The wound left is immediately closed, the margins being undercut if this is necessary to approximation.

If veins are exposed after the skin has been removed, it is better to ligate and excise them before sewing up. The removed piece of skin is held in a piece of gauze, deep surface upwards, and then with curved scissors every

particle of adhering fat is cut away. Holes are made in it with a punch to prevent accumulation of fluid beneath it. It is then applied to the granulating wound, prepared as for other methods of grafting, and sutured in place. It may be stretched somewhat, and it must be kept closely applied by a dressing of gauze and wool firmly bandaged on.

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**WRITER'S CRAMP** (see NEUROSES, OCCUPATION).

**WRY-NECK** (see TORTICOLLIS AND WRY-NECK).

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**XANTHELASMA** (see XANTHOMA).

**XANTHOMA.**—Three types of xanthoma, or xanthelasma (Gr. *ξανθός*, yellow), are generally recognized in the literature: (1) *Xanthoma planum*, flat or yellow spots or plaques of the colour and consistence of natural chamois leather, occurring commonly in the loose skin of the eyelids. (2) *Xanthoma papulosum* or *tuberosum*, papular or nodular elevations of a yellowish or reddish colour, but revealing their true nature on diascopy. (3) An exceedingly rare variety which results from confluence of the elements of the second variety, and is called by Darier *xanthome en tumeurs*. All three varieties, which can occur separately or together, are of a yellowish colour and of a consistence which varies with the amount of fibrous tissue present. They closely resemble one another in their histological characters.

**Etiology.**—The causation of the disease is still *sub judice*, and it is in dispute whether it should be classed among the tumours or the granulomata. There is little doubt that, among the affected, diabetes, glycosuria, and diseases of the liver, especially those producing cholæmia, are common, and every patient with xanthoma should be examined from these points of view. It has been established that the blood of patients with any of the above complaints (including xanthoma) is rich in cholesterin, and many investigators have assumed that the pathological pigment found in

xanthoma lesions is a liquid cholesterin ester derived from an excess of this substance in the blood and tissue fluids.

**Histopathology.**—The pronounced yellow colour of the xanthoma lesions is due to the presence of patches of peculiar pigment in the cutis. Under the microscope this may be identified as fine pointed crystals or granules, staining a brilliant refractile orange colour with Sudan III and readily soluble in alcohol or ether. The crystals and amorphous granules melt at a temperature a little above blood heat, and other chemical reactions justify their classification among the fatty acids or esters. Associated with the pigment, and containing particles of it, are large, round connective-tissue cells grouped mainly round the smaller blood-vessels. These are the so-called xanthoma cells. They are rich in protoplasm, and are occasionally multinucleated like the giant cells of a granuloma.

**Symptomatology.**—The onset of *xanthoma of the eyelids* is very gradual, often unnoticed, and there are usually no subjective symptoms. The bright yellow plaques are often bilaterally and symmetrically situated at the inner canthi, and after reaching a certain size tend to persist indefinitely, without any disturbance of the general health. Darier notes an occasional association with hepatic cirrhosis, and states that it may be combined with the papulonodular variety. *Xanthoma tuberosum multiplex* may be looked upon as a generalized

## XANTHOMA

variety of the palpebral and nodular types; the eyelids may or may not be affected. It is characterized by the development, at intervals, of crops of xanthomatous papules and nodules tending to affect the skin over bony prominences, the knees, the knuckles, and the hairy scalp. Pale xanthomatous streaks appear sometimes in the flexures and on the palms and soles. *Xanthoma diabeticorum* is a very rare acute condition, and usually reaches full development in about a month. The lesions are pink or yellowish follicular papules, general in distribution and evanescent in duration. Intense pruritus is a constant feature of this type of xanthoma.

Xanthomatization is a secondary phenomenon akin to lichenification, which has been described under PRURIGO, and, like it, may ensue as a result of scratching in predisposed individuals.

**Diagnosis.**—The palpebral form is most unlikely to be mistaken for any other condition. Its specific colour and consistence are sufficient to distinguish it from *adenomatous and milium cysts*, which are very common about the eyelids and nose. An exceedingly rare condition termed *pseudoxanthoma elasticum*, the histological basis of which is a spontaneous destruction of elastic tissue (elastorrhexis), with thickening and discoloration of the involved skin, has nothing in common with true xanthoma, which, once seen, can scarcely be mistaken for any other disease.

**Treatment.**—When there is marked chloemia the treatment must be directed to improvement of the hepatic functions, and glycosuria and diabetes, when present, must be treated on the usual lines. X-rays may be expected to do good in the nodular and tuberculous varieties on the trunk and limbs, especially in those in which young fibrous tissue is a prominent element of the structure. For the palpebral variety I have found acupuncture with Unna's microbrenner very successful. This little instrument supplies just that amount of heat necessary to cause melting of the lipid granules, and the alteration in physical characters thus brought about seems to lead to their absorption. A very small area should be treated at each sitting, as the resulting oedema of the eyelid is unsightly and may alarm a nervous patient. The procedure is only slightly painful.

HENRY SEMON.

**XERODERMIA** (see ICHTHYOSIS).

## X-RAYS, DIAGNOSTIC USES OF

**XERODERMIA PIGMENTOSUM** (*syn.* Kaposi's Disease).—This rare condition begins in early life with freckling, telangiectases, and warty growths; as a rule, atrophic spots also develop. Some of the lesions may undergo carcinomatous change. In view of this possibility, during the pre-carcinomatous stage attempts should be made to avoid irritation from sunlight. Once the malignant change has occurred, measures similar to those employed in carcinoma of the skin (see SKIN, MALIGNANT GROWTHS OF) are demanded.

H. MACCORMAC.

**XEROSTOMA** (see SALIVA, ANOMALIES OF SECRETION OF).

**X-RAY DERMATITIS** (see DERMATITIS, X-RAY).

**X-RAYS, DIAGNOSTIC USES OF.**—The improvements which have been made during the past few years in apparatus and technique, together with the advancement of knowledge in the interpretation of radiographic appearances, have resulted in a very wide extension of the field in which this means of investigation can usefully be employed. To obtain the maximum benefit from radiography, however, it must constantly be borne in mind that the evidence thus obtained should be viewed in its proper perspective in relation to other methods of examination; and it is the duty of the radiologist not only to make a correct interpretation of the radiographic findings, but also to assess the degree of prominence which should be accorded to these findings in any particular case. For example, a fracture or dislocation in almost any part of the body can be demonstrated or excluded with absolute certainty, but the failure to demonstrate biliary calculi does not by any means exclude the possibility of their presence.

Apart from these considerations, the value of a radiographic examination will depend very largely upon the method of conducting it. First, it cannot be too strongly emphasized that screen examination alone is utterly insufficient for the formation of a useful opinion in practically any condition; even gross lesions of large and superficial bones may be (and frequently are) quite indistinguishable on the screen. In the investigation of the alimentary and respiratory systems, screen examination plays a most important part, but even here plates of good quality should also be obtained.

In the second place, the plates obtained



## X-RAYS, DIAGNOSTIC USES OF

must be of satisfactory quality, the result of correct exposure and development and of complete elimination of movement on the part of the patient.

Thirdly, it is of the utmost importance that the view obtained be one with which the operator is thoroughly familiar; every radiogram should therefore be secured according to a definite rule, which fixes the position of the part under examination, the plate, and the central radiation from the tube. In this way only is it possible to reproduce exactly identical views of any portion of the normal body, thereby rendering comparatively easy of recognition any departure from the normal which may be present.

Finally, wherever possible, two views at right angles should always be obtained. In situations where this is impracticable (e.g. the hip, shoulder, lumbar spine, etc.) stereoscopic radiograms are frequently of great value.

### I. SKELETAL SYSTEM

A satisfactory radiogram will show the lamellar structure of any normal bone examined; in the long bones clear differentiation should be obtained of the medullary canal, the compact and the cancellous bony tissue. Absorption of a certain proportion of the lamellæ takes place as the individual advances in age, with resultant general rarefaction of both cortical and cancellous bone as seen on the radiogram; the medullary canal is also increased in width. Very similar changes, either general or local, are seen as the result of disuse.

For the correct interpretation of radiograms of young subjects a thorough knowledge of the process of ossification must be acquired from anatomical textbooks and personal observation; this statement especially applies to the time of appearance of the ossific centres in the various epiphyses, and of the union of the epiphyses with the diaphyses.

#### INFECTIVE AND TOXIC BONE CONDITIONS

**1. Pyogenic infections.**—It must be clearly recognized that during the first few days of an acute infection no abnormality can be made out; later, the affected part of the bone shows rarefaction, which may go on to complete loss of bone shadow (abscess-formation). There is always some blurring of the bony structure, but this is rarely so complete as in tuberculous infections. If the shaft of a bone is affected, the periosteum is frequently seen to be thickened

and raised from the surface of the bone. If a articulation is involved, destruction of the articular cartilage eventually allows the bone extremities to become more closely approximated than is normal, while their irregularity of outline and structure is generally early apparent as evidence of erosion and necrosis of bone (PLATE 42, Figs. 1, 2.)

In chronic infections of this nature, irregular sclerosis and new periosteal bone-formation are the most characteristic signs. Necrosis shown by superficial irregularity with complete loss of structure. Sequestra commonly show greatly increased opacity, and are seen to be separated from the surrounding bone. Chronic abscesses are recognized as translucent areas the bony walls of which are sharp in outline and may be increased in density. Healing of the lesion is indicated by clearer definition of bony structure. In the joints a greater or less degree of bony continuity is frequently established across the articulation.

**2. Pneumococcal infections.**—The changes differ generally from those just described by being more localized and showing less activity and loss of bony structure. Periosteal "blisters" with but slight affection of the underlying shaft are common in the long bones.

**3. Typhoid infections.**—Acute typhoid osteomyelitis may be indistinguishable from pyogenic infection. The common post-typhoid lesion shows a localized chronic periostitis, often with an abscess cavity in the new subperiosteal bone.

**4. Tuberculous infections.**—These are characterized by extreme loss of definition of bony structure, giving a typical "woolly" appearance, which often extends for a considerable distance beyond the more obvious focus of disease. Necrosis and abscess-formation, resulting in great destruction of bone, are generally seen in lesions of any considerable duration. In the shafts of the long bones it is not uncommon to find central destructive changes of this nature associated with an extensive subperiosteal new-bone formation producing great enlargement of the shaft (spina ventosa). An atypical chronic periostitis may sometimes afford the only evidence of a bony lesion. In children tuberculous lesions usually affect the ends of the diaphyses.

When a joint is involved, widespread destruction of the articular surfaces is common but the disease is sometimes first manifested on the diaphyseal side of the epiphyseal line.





Fig. 1.—Chronic pyogenic osteomyelitis of tibia.



Fig. 2.—Chronic pyogenic arthritis of hip and osteomyelitis of femur.



Fig. 3.—Tuberculosis of knee-joint.



Fig. 4.—Syphilitic osteitis of fibula.



Fig. 5.—Rheumatoid arthritis.

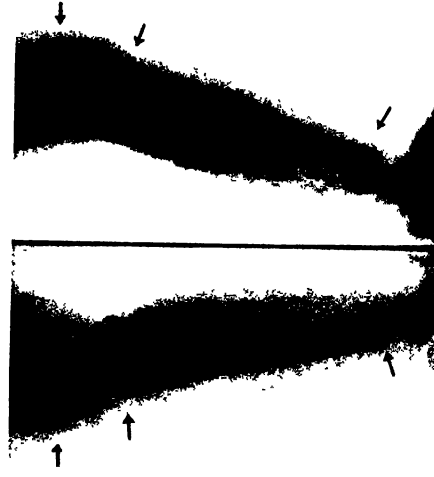


Fig. 6.—Rickets.

## X-RAYS, DIAGNOSTIC USES OF

early "synovial tuberculosis" definite X-ray diagnosis is impossible; there is merely an increased translucency of the bones entering into the articulation.

In old tuberculous lesions sclerosis is not so marked as in chronic pyogenic infections, but the destruction of bone is generally seen to have been greater than in the latter condition (PLATE 42, Fig. 3).

**5. Syphilitic infections.**—Gumma of bone causes a complete localized bony destruction, very similar in its X-ray appearances to the destruction caused by an endosteal sarcoma. In the case of a gumma, however, the changes are commonly more localized, and do not extend so far in the central portion of the bone; there is nearly always evidence of inflammatory reaction in the form of periostitis. Diffuse gummatous infiltration produces widespread irregular destructive changes combined with sclerosis and periostitis (PLATE 42, Fig. 4).

Chronic syphilitic inflammation is shown by diffuse sclerosis and hyperostosis, generally less irregular in its distribution than in pyogenic infections, and by encroachment upon or apparent obliteration of the medullary canal by sclerosed bone. This latter point is of great significance in the diagnosis of syphilitic from other chronic inflammations of bone.

**6. Gonorrhœal infections.**—Cases of non-suppurative gonorrhœal arthritis show great rarefaction and irregularity of bony structure, confined to the neighbourhood of the articular extremities. The articular surfaces of the bones present no irregularity.

The suppurative forms of gonorrhœal arthritis cannot be distinguished by X-ray examination alone from other lesions of pyogenic origin.

### **7. Polyarticular rheumatoid arthritis.**

--In comparatively early cases there is no radiographic abnormality except slight general rarefaction. In more advanced cases the following changes will be seen in the affected joints: (a) The distinction between the thin layer of compact bone covering the articular surfaces, and the underlying cancellous bone is largely lost. The articular surfaces also show a fine irregularity. These changes, when occurring in the carpus, render it difficult or even impossible to distinguish the outlines of the individual bones. (b) Small round translucent areas, with clear-cut margins, are seen; this gives a "punched-out" appearance which is typical of the affection. Small bony outgrowths may be seen at the margins of the articular surfaces, but these are spiky, irregu-

lar, and fragile in appearance, quite unlike the smooth, well-formed osteophytes of osteoarthritis. (PLATE 42, Fig. 5.) In very advanced cases of rheumatoid arthritis the X-ray appearances closely simulate those of tuberculosis.

**8. Still's disease.**—The radiographic joint-changes in this condition are similar to those observed in rheumatoid arthritis (*see above*), but the destructive changes are generally less advanced. In early cases it will be seen that, in conjunction with the increased translucency, the carpus shows advanced ossification when compared with the carpus of a normal child of the same age. This advanced ossification is not pathognomonic of Still's disease, but is frequently observed in other chronic inflammatory affections of growing bones and ununited epiphyses.

**9. Hypertrophic pulmonary osteoarthropathy.**—The clubbing of the fingers seen in this and in other diseases is not found to be associated with bony change. The metacarpals and metatarsals, with the first one or two rows of phalanges, show rarefaction, with a layer of subperiosteal new-bone formation. This change may also sometimes be seen in the lower ends of the long bones of the limbs, and, as described above, advanced ossification of the carpus and tarsus may take place in young subjects.

**10. Pseudo-coxalgia** (Perthes' disease; osteochondritis juvenilis).—The X-ray appearances are very similar to those of tuberculosis. There is usually marked irregularity of the head of the femur and of the epiphyseal cartilage between it and the neck. Destructive changes are commonly observed in the upper part of the neck, and also sometimes in the acetabulum; and the growth of the entire pelvic girdle is generally retarded upon the affected side. The bones do not present the blurring of structure which is so characteristic of tuberculosis.

## SYSTEMIC DISEASES AFFECTING BONE

**1. Rickets.**—The typical appearances of this disease are best seen in the long bones. The shaft shows rarefaction of the cortex with undue width of the medullary canal. The rarefaction may give rise to localized cystic appearances, and spontaneous fractures are fairly common; they unite readily. The periosteum can often be seen as a thickened layer. When curvature has occurred, subperiosteal new bone is laid down in the concavity of the curve.

## X-RAYS, DIAGNOSTIC USES OF

The regions of the epiphyses also present characteristic changes. The epiphysis itself shows retarded growth. The extremity of the diaphysis is greatly expanded, so that it overlaps the epiphysis to a quite abnormal extent; while owing to the disordered process of ossification the opposing surfaces of both epiphysis and diaphysis are ragged and irregular (PLATE 42, Fig. 6). In bones other than the long bones (e.g. the pelvis, the spine), the disease is only manifest by the deformities which it produces; the characteristic bony changes cannot be made out. As the disease subsides the bones become abnormally dense.

**2. Scurvy.**—The X-ray diagnosis of this disease depends upon the recognition of subperiosteal hæmorrhages; apart from these, the bones only show some degree of rarefaction and absorption which presents no typical features. When quite recent, the hæmorrhages may be unrecognizable, but later the deposition of calcium salts results in a fusiform shadow of fairly uniform opacity, and devoid of definite structure, which is unmistakable. The thickened periosteum can generally be seen limiting this opacity. There is no erosion of the underlying bone. It must be remembered that children who are the subject of this disease are frequently rachitic, and in these cases the appearances characteristic of both affections will be present.

**3. Gout.**—Joints which are affected by gouty arthritis present no typical X-ray appearances. The changes shown may be similar to those seen in osteo-arthritis or in rheumatoid arthritis—more commonly the latter.

### BONE DISEASES OF DOUBTFUL ORIGIN

**1. Acromegaly.**—Examination of the skull provides evidence of a pituitary tumour in a certain proportion of cases of this disease. The sella turcica may be simply expanded, and thus appear unduly large, or the bony walls may show actual erosion, in which case the posterior clinoid processes are first involved.

The enlargement of bone which constitutes a characteristic symptom in acromegaly is seen to be due to a simple overgrowth of the cancellous tissue, without sclerosis or other modification in structure. The clubbing of the fingers is partly accounted for by accentuation of the normal bulbous ends of the terminal phalanges.

In *leontiasis ossea*, which may, perhaps, be tentatively placed in the same category as acromegaly, the affected bones are again seen to be the subjects of a simple hypertrophy.

**2. Fragilitas ossium** (osteogenesis imperfecta).—The cortical layer of bone is thin and the cancellous layer broader than in normal bone; the cancellous tissue, moreover shows rarefaction and diffuse absorption of lamellæ. Cystic appearances are sometimes present. The occurrence of multiple fracture renders the diagnosis easy. Bony union occurs rapidly, but generally with considerable deformity.

**3. Osteitis deformans.**—The characteristic X-ray appearances of this disease are generally best seen in the tibia and femur, although other bones—notably those of the skull, pelvis, and spine—are usually affected to a marked degree. In a patient who has suffered from the disease for some time the bones are seen to be increased in thickness, curved, and devoid of all differentiation in structure, so that they throw a shadow of uniform density throughout their extent sometimes varied by cystic-looking spaces. The medullary canal is not readily distinguishable. Sometimes spontaneous fractures occur (PLATE 43, Fig. 1.)

**4. Osteitis fibrosa.**—In this disease the radiogram shows that the affected bone or bones are broadened, often increased in length, and distorted. The replacement of bone by longitudinal striæ of fibrous tissue results in a typical openwork appearance, in which all differentiation between cortical and cancellous tissue is lost. Local cyst-formation and spontaneous fractures are common. (PLATE 43, Fig. 2.)

**5. Osteo - arthritis** (hypertrophic osteo-arthritis). In very early cases no X-ray changes are discernible. Later, there is some condensation of the cortical layer covering the articular surfaces, and by the time this is well marked, osteophytic formation has generally taken place. The osteophytes are seen at the margins of the articular surfaces, and show the structure of the cortical bone. They are regular in outline, smooth, and often rounded (*cf.* Rheumatoid Arthritis, p. 547). In an advanced case the articular surfaces are more closely approximated than normally, and the cortical bone is often seen to be very dense and sometimes grooved by pressure. Ossification may extend into the ligaments, and calcified or ossified bodies are often seen lying in the joint cavity. (PLATE 43, Fig. 3.) When this disease attacks the vertebræ (spondylitis deformans), extension of ossification into the ligaments often results in bony ankylosis between the bodies of adjacent vertebræ

## X-RAYS, DIAGNOSTIC USES OF

(PLATE 43, Fig. 4). Osteo-arthritis, with this one exception, does not produce bony ankylosis.

**6. Osteomalacia.**—In children and adults the shafts of the long bones are narrowed and show great rarefaction. The cortical layer is very thin and the medullary canal unduly wide, owing to the absorption of cancellous bone. Curvature and spontaneous fracture are common. The extremities of the long bones are expanded and share in the general rarefaction; the pelvis and spine show great rarefaction, and, frequently, characteristic deformities. If this disease actually occurs in infants (which appears doubtful) it is indistinguishable radiographically from *fragilitas ossium*.

**7. Achondroplasia.**—In infancy the extremities of the diaphyses of the long bones of the limbs are smooth and abnormally dense. They are frequently broader than usual, owing to excessive formation of subperiosteal new bone, and thus may present a superficial resemblance to rickets; but there is no evidence of ossification or irregularity in the epiphyseal cartilage, and the epiphysis itself is unaffected (PLATE 43, Fig. 5). In children about the age of puberty all the epiphyses may be united, but this does not occur in all cases. The long bones of the limbs are short and deformed, and the articular extremities often present great irregularity (PLATE 43, Fig. 6).

### DEVELOPMENTAL ABNORMALITIES OF BONE

Absence, reduplication, malformation, and local gigantism are sufficiently obvious on X-ray examination. Small additional ossicles in the carpus and tarsus require careful recognition, as they may simulate fractures; in the carpus the *os triangulare*, lying below the styloid process of the ulna, and the *os centrale*, lying on the dorsum between the scaphoid, trapezium, and *os magnum*, are both fairly often seen. In the tarsus the *os trigonum tarsi*, lying behind the postero-internal margin of the astragalus, is very frequently present. A separate centre of ossification for the tubercle of the navicular is sometimes found, and the two parts of the bone may remain separate throughout life.

These abnormalities are often bilateral—a point of some diagnostic importance.

Congenital dislocation may occur in the hip, shoulder, knee, and inferior radio-ulnar joints, but the hip is the only situation in which this condition is anything but a rarity. In examples of this abnormality the head of the femur is displaced upwards and is often de-

formed, while the neck of the bone is distorted, and the acetabulum is poorly formed, or almost absent. The condition is frequently bilateral. Minor degrees of displacement and deformity are recognized by continuing the curve formed by the lower border of the neck of the femur, and the curve formed by the upper border of the obturator foramen. The imaginary lines prolonging these two curves should become directly continuous. If they meet at an angle there is displacement of the head of the femur or some other deformity of the parts—e.g. *coxa vara*.

Half-vertebræ, which in the dorsal region may or may not bear a rib, are readily recognized, but here as elsewhere a radiogram of good quality is essential, otherwise the condition may be mistaken for localized tuberculosis with unilateral collapse of a single vertebra.

Cervical ribs are comparatively common abnormalities. They are generally bilateral, though the rib is often much larger on one side. In Sprengel's shoulder the abnormal bone-formation can be demonstrated.

### NEW GROWTHS OF BONE

**1. Benign.**—The only benign new growths of bone which are seen with any frequency are chondromata (enchondromata), and osteomata. The *enchondroma* produces a fairly symmetrical expansion of the bone in which it occurs, accompanied by actual absorption. The radiogram shows a central cystic appearance surrounded by an expanded shell of bone. There are no inflammatory changes in the bone, and thinning is always accompanied by expansion. The tumour possesses a definite clear-cut margin. Diagnosis from cysts cannot be made by radiography alone. (PLATE 44, Fig. 1.)

*Osteomata* may be cancellous or compact (ivory). Those of the cancellous variety show the normal structure of this type of bone, and are seen to be sessile or pedunculated in their attachment to the parent bone. The surface of the osteoma may show some superficial rarefaction, or necrosis, as a result of pressure, but otherwise the tumour is perfectly regular in structure, as is the bone from which it springs. Compact or ivory osteomata occur only in relation with the bones of the skull. They throw a very dense shadow, regular in outline and consistence. Usually there is no change in the underlying bone, but these tumours sometimes separate spontaneously owing to the density of the growth obstructing its own blood supply; when this is

## X-RAYS, DIAGNOSTIC USES OF

occurring, rarefaction and necrosis may be seen at the point of attachment.

**2. Malignant.**—Endosteal and periosteal sarcoma are fairly common, and present characteristic appearances. In the *endosteal* (round or spindle-celled) *sarcoma* the radiogram shows a central destruction, which at a later stage involves the entire width of the bone. There is no new-bone formation, no expansion, no inflammatory change. Erosion takes place without any preceding loss of detail in the bony structure, and spicules of bone retaining perfect detail may be seen projecting into the area occupied by the growth. No margin to the changes can be defined; spontaneous fracture is common. Diagnosis from simple cysts and myelomata is made by the absence of expansion of the surrounding bony walls, and the ill-defined limits of the destructive changes; and from inflammatory conditions by the absence of periostitis, new-bone formation, or blurring of detail in the contiguous bony structure.

In *periosteal sarcoma* the radiogram shows a mass of delicate, almost feathery bony spicules projecting at right angles to the surface of the affected bone. These spicules, both in their axial relation to the parent bone and in their fragile structure, are quite different from the new-bone formation of any other condition. In early cases the underlying bone shows a little erosion of the same nature as that seen in endosteal sarcoma; later, the destructive changes become much more marked, but are rarely so advanced as in the central neoplasm. Spontaneous fracture, however, is not uncommon. (PLATE 44, Fig. 2.)

*Myeloid sarcoma*, or *myeloma*, presents on radiographic examination a central translucent area surrounded by thinned and expanded bony walls. The margins of the central tumour are always perfectly clear-cut and well defined. The expansion may become very great before the bony wall is completely destroyed at one or more points. Spontaneous fracture is often seen. There is no new-bone formation apart from that which accounts for the expansion, nor are there inflammatory changes. The appearances closely simulate those of bone-cysts, and differential diagnosis from the latter condition by radiographic means is frequently impossible. In general it may be said that a myeloma tends to expand the bone more or less equally in all directions, while a cyst often involves a considerable length of the shaft of the bone without producing much lateral ex-

pansion. (PLATE 44, Fig. 3.) The diagnosis from round- or spindle-celled endosteal sarcoma is generally easy (see above).

*Primary carcinoma* does not occur in bone the cases which have been cited as example of this condition being probably secondary to some inconspicuous growth, often in the prostate or thyroid. *Secondary carcinomatous deposits* are fairly common; they are most frequently found in the upper end of the humerus, the neck of the femur, the spinal column, and the skull. The usual radiographic appearance produced by these deposits is very similar to that of an endosteal sarcoma; generally speaking, carcinoma tends to produce a rather more diffuse change, several small areas of disease being present, separated by more or less normal bone (PLATE 44, Fig. 4). In other cases where the secondary deposit is of long standing, and has grown slowly, actual expansion of bone may be seen, and at times sclerosis; and these appearances give rise to great difficulties in diagnosis. In the body of a vertebra, also, it may be impossible to make out the nature of the bony change, the radiographic finding being simply that of collapse of a vertebra. In all these cases careful clinical examination is necessary to complete the diagnosis.

**3. Cysts of bone.**—Many diseases—e.g. rickets, osteitis deformans, osteitis fibrosa—give rise to radiographic appearances which are commonly spoken of as "cystic." The term bone-cyst should, however, be confined to those conditions where an actual cavity, containing fluid or semi-fluid material, exists in the bone. The pathology of most of these cysts is obscure; some are probably inflammatory, others may result from the liquefaction of myelomata or chondromata. The radiographic appearances are almost identical with those of myeloma, and the differential diagnosis has been discussed under that heading.

*Hydatid cysts of bone* are multilocular. In the long bones they cannot be differentiated radiographically from other cysts. When they occur in the vertebral bodies the radiogram generally shows a simple collapse, often confined to a single vertebra, and similar to that seen in carcinomatous deposits.

### STATIC CHANGES IN BONE

Static deformities are recognized by the absence of any alteration in structure indicative of past or present bony injury or disease. The diagnosis, as excluding any such injury or



Fig. 1.—Osteitis deformans.

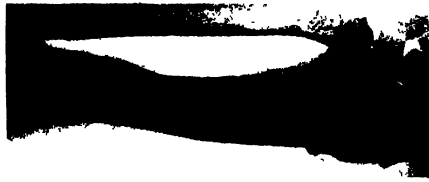


Fig. 2.—Osteitis fibrosa.



Fig. 3.—Osteo-arthritis of knee;  
opaque bodies in joint.



Fig. 4.—Spondylitis deformans.



Fig. 5.—Achondroplasia in an infant.



Fig. 6.—Achondroplasia in a child. 15.





Fig. 1.—Multiple enchondromata.

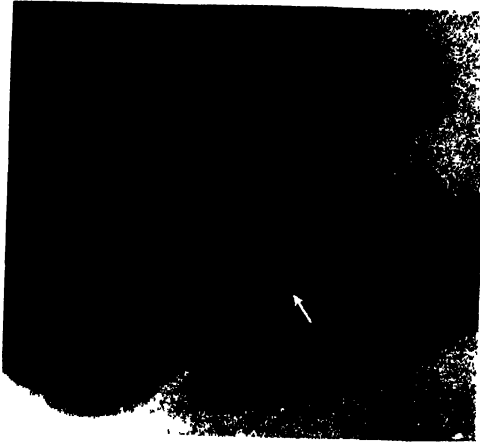


Fig. 2.—Periosteal sarcoma of ilium.



Fig. 3.—Myeloma of ulna.



Fig. 4.—Secondary carcinoma of upper end of femur and adjacent pelvic bones, with spontaneous fracture.



Fig. 5.—Hydropericardium.



Fig. 6.—Empyema of right maxillary antrum.

## X-RAYS, DIAGNOSTIC USES OF

disease, is frequently of great importance, but is generally easy.

### II. MUSCLES AND FASCIÆ

In a radiogram of good quality the outlines of the large superficial muscles, with their tendons of insertion, are generally discernible, and **rupture** of such structures as the biceps in the upper limb and the quadriceps extensor tendon in the lower can often be recognized. The smaller and more deeply placed muscles, however, rarely produce a distinctive shadow.

**Localized ossification**, apart from any preceding injury, is not infrequently found in fasciæ and intermuscular septa. The most important example of this condition is the calcaneal spur. These spurs may attain considerable dimensions, are frequently bilateral, and are sometimes fractured as a result of comparatively slight degrees of trauma. They are most commonly found in middle-aged subjects, but may occur in children.

**Myositis ossificans** is readily recognized by the diffuse formation of bone in the muscles. Since the muscles first involved are those of the back, however, care is necessary in early cases to distinguish the new-bone formation from the superimposed shadows which are thrown by the normal parts.

Traumatic myositis ossificans is a fairly common sequela of bony injury, especially when the injury has been slight; a radiogram will show new bone-formation in the soft tissues, completely separated from the shaft of the bone. The new bone is fairly regular in structure, and the lamellæ are arranged parallel to the shaft. The shaft may show evidence of injury, but no destructive changes.

Of the parasitic diseases which may affect muscles, **trichinelliasis** is of importance from the radiographic point of view, since the trichinellæ, when calcified, throw characteristic shadows. These are always multiple, vary in shape from round to fusiform (according to the relation of the long axis of the trichinella to the axis of radiation), and are about  $\frac{1}{4}$  in. by  $\frac{1}{2}$  in. in size. The calcification is seen to be more advanced at the ends of the worm.

### III. CARDIO-VASCULAR SYSTEM

Both screen and plate examination should be employed in the investigation of the heart and blood-vessels, and, unless the condition of the patient precludes it, the upright position should be adopted.

The screen examination is carried out as follows: The patient is placed facing the operator, and a general survey is made of the whole thorax; the diaphragms of the tube-box are then closed so as to allow of a small area of illumination only around the shadows of the heart and great vessels. The patient is now slowly rotated into the right oblique position, returned to his original posture, and slowly rotated into the left oblique position. In this way the size, shape, position, and movement of nearly the whole of the heart can be made out, and the ascending and transverse portions of the aortic arch inspected. (The shadow of the normal descending thoracic aorta is overlapped and obscured by those of other structures.)

Radiograms should be secured in any position in which the screen appearances suggest abnormality.

*Size of the heart.*—To determine this it is necessary to employ orthodiagraphy, or, as is far more convenient, to have the X-ray tube at a distance of 6 ft. from the patient, thereby rendering negligible the distortion caused by the divergence of the radiations.

The size of the heart varies greatly in normal subjects, but maintains a fairly constant proportion to the size of the bony thorax. The greatest transverse diameter of the heart is 39-50 per cent. of the greatest transverse diameter of the bony thorax. When the percentage reaches 53 or more definite pathological enlargement can be diagnosed. The measurements should be made from plates taken at mid-inspiration in the upright position (C. S. Danzer).

It is generally possible to state which side of the heart is more involved in enlargement, but dilatation cannot be differentiated from hypertrophy with any degree of certainty by radiographic means alone.

The *shape of the heart* in a normal subject varies with that of the thorax. Dilatation or hypertrophy of any of the chambers will cause a corresponding modification in the normal outline, but the descriptions which have been given of alterations in shape characteristic of various lesions require further confirmation.

**Aneurysm of the heart** produces a localized extension of the cardiac shadow, less dense than the shadow of the main organ, and commonly showing expansile pulsation.

It is of the utmost importance to note that in all general or local enlargements of the

## X-RAYS, DIAGNOSTIC USES OF

heart itself, the right border of the viscus (formed by the right auricle and ventricle) makes an acute angle with the right leaf of the diaphragm (cardio-phrenic angle). In pericardial effusion this angle is obliterated, and the cardiac shadow becomes enlarged, globular in shape, and sometimes devoid of movement (PLATE 44, Fig. 5).

**Aneurysm of the aorta** causes an enlargement of the shadow normally produced by that vessel. The enlargement may be localized or diffuse, corresponding with the saccular or fusiform nature of the aneurysm, and may involve any part of the aorta. In this connexion attention must be called to the normal extension to the left of the aortic shadow at the junction of the ascending and transverse portions of the arch. Failure to recognize this as a normal anatomical condition has resulted in many mistaken diagnoses of aneurysm.

Aneurysm must be distinguished from *enlarged glands*, and this is often a matter of great difficulty. The following points should be noted in considering the nature of an abnormal shadow connected with that of the aorta :

1. Aneurysm tends to be unilateral; enlarged glands are nearly always bilateral.
2. Expansile pulsation is sometimes seen in an aneurysm; pulsation produced in glands by their approximation to the heart or vessels is never expansile.
3. In aneurysm some part of the abnormal shadow always appears directly continuous with that of the aorta; in the case of enlarged glands an unbroken line of demarcation from the aorta may be made out.
4. The left border of the heart is often abnormally horizontal in position when aneurysm is present. This point is not of much practical value.

It should be recognized that in some cases the diagnosis of aneurysm may present one of the most difficult problems that the radiologist is called upon to solve.

**Aneurysms of peripheral vessels.**—Large aneurysms of superficial peripheral vessels can sometimes be seen on X-ray examination, but a definite diagnosis can rarely be made by this means.

Calcification of peripheral vessels is readily demonstrated, and this observation is sometimes of value to the surgeon in connexion with the treatment of senile gangrene and allied disorders.

## IV. RESPIRATORY SYSTEM

1. *Nasal fossæ and accessory sinuses.*—In a postero-anterior radiogram of the skull the nasal fossæ are shown as a pear-shaped translucent area, modified by the opacities of the nasal septum and the middle and inferior turbinate bones. Obstruction of one or both nasal fossæ produces opacity in the corresponding portion of the radiogram. Deviation of the septum and hypertrophy of the turbinates can also be demonstrated.

X-ray examination of this region, however, is most usually sought in connexion with the condition of the accessory nasal sinuses. These are normally translucent to radiation in virtue of the air which they contain; when the air is displaced by fluid (e.g. pus or blood) or by tumour-formation the normal translucency is lost. Oedema of the mucosa lining the sinuses, without actual collection of fluid, produces a lesser degree of opacity.

The condition of the frontal and ethmoidal sinuses and the maxillary antra are best studied in a postero-anterior radiogram, as it is then possible to compare the translucency of the corresponding sinuses on the two sides. It must be remembered, however, that bilateral affections are by no means uncommon. The sphenoidal sinuses can rarely be well seen in the postero-anterior radiogram, but are readily distinguished in a lateral view of the skull. Minor degrees of opacity of the sphenoidal sinuses are not easy to recognize. The frontal sinuses are seen to present great variations in size and shape, and frequently to be asymmetrical. Sometimes the sinus on one or both sides is seen to be absent or quite rudimentary; this condition should not be confused with *opacity* of an existing sinus, since in the latter the outlines of the opaque sinus can always be distinguished.

The maxillary antra are usually symmetrical, but vary in size considerably in different subjects. No useful comparison of the translucency of the antra can be made unless the radiogram obtained is an exact postero-anterior view; any deviation from this, due to rotation of the head or inaccurate centring of the tube will give rise to apparent inequality in the translucency of the antra, owing to the asymmetrical distribution of the superimposed shadows. Fig. 6, PLATE 44, shows an opaque antrum due to **empyema**.

The ethmoidal sinuses when diseased generally show a moderate increase of opacity, together with some irregularity of structure

## X-RAYS, DIAGNOSTIC USES OF

due to erosion of the bony walls enclosing the small air-cells.

2. *The larynx, trachea, and bronchi.*—The larynx and the cervical portion of the trachea can be readily differentiated from surrounding tissues owing to their translucency to radiation in virtue of the air which they normally contain. Displacement of these structures and the presence of opaque foreign bodies therein can easily be demonstrated. Calcification of the laryngeal cartilages is common after middle age, and produces irregular granular opacities. The intrathoracic portion of the trachea and the bronchi cannot generally be clearly seen.

**Dilatation of a bronchus** (bronchiectasis) produces rounded or fusiform areas, the translucency of which may be increased or diminished, according to the contents of the dilatation (air or fluid). If a large bronchiectatic cavity be examined in the upright position an upper translucent and a lower opaque area may be seen, the demarcation between the two being formed by the horizontal surface of the contained fluid.

Bronchiectatic cavities are generally multiple, and are accompanied by fibrosis of the surrounding lung.

3. *The lungs and pleura.*—In the X-ray investigation of these structures screen examination plays a most important part, but one or more plates taken with the breath held at the end of the inspiration should always be obtained subsequently. It is frequently advantageous to examine the patient both in the supine and in the vertical positions when this is possible.

The preliminary screen examination should include the whole thorax; the diaphragm of the tube-box is then closed so as to limit the illumination to a transverse slit; this facilitates comparison of corresponding areas on the two sides of the thorax.

Attention should first be directed to the apices, and any inequality in translucency duly noted. Here, and in the subsequent examination of other regions of the thorax, the patient should be observed during both normal and forced respiration. Inspiration produces "lighting-up," or increase of translucency, in a normal lung, and any diminution of this lighting-up is indicative of deficient air-entry. The lighting-up in the right and left apices should be equal in degree, or only slightly more marked on the right side. A similar examination is made of all other parts of the two lungs, special attention being paid to any

area which appears abnormal. When at the end of this systematic examination the diaphragm is brought into the field of illumination, the shape, position, and movements of this structure on normal and on forced respiration must be carefully investigated.

In normal subjects the position of the diaphragm undergoes constant variations with the respiratory movements, the usual degrees of excursion being about  $\frac{1}{2}$  in. in quiet and 2–2 $\frac{1}{2}$  in. in forced respiration. Variations in position are also produced by the condition of the stomach and intestines. Generally speaking, the upper limit of the right leaf of the diaphragm corresponds to the fourth costal cartilage in front, while the left leaf lies  $\frac{1}{2}$ –1 $\frac{1}{2}$  in. lower.

The radiographic appearances of the lung structure can only be studied in a radiogram taken with the breath held; complete immobilization is absolutely essential. It will be seen that the normal lung presents numerous striate opacities, radiating from the hilus and becoming finer and more diffuse as the periphery of the organ is approached. These striæ are produced by the bronchi and the larger bronchioli. The translucency of the lung tissue is further modified by the shadows of the smaller bronchioli the long axes of which present every variety of angle to the axis of radiation; these produce the uniform fine variation in light and shade which is known as the "normal lung mottling."

4. *Thoracic lymphatic glands.*—A certain degree of enlargement of the bronchial glands commonly takes place very early in life, and persists or becomes accentuated as age increases. Only very considerable degrees of enlargement can be looked upon as abnormal. The aortic and posterior mediastinal glands, on the other hand, are not often noticeably enlarged to X-ray observation except as the result of some definite pathological condition—most commonly a new growth.

**Emphysema.**—The transverse diameter of the thorax is seen to be increased in proportion to the vertical. The lung tissue is abnormally translucent, but shows very little lighting-up on inspiration. The respiratory movements of the diaphragm are increased, and those of the bony thorax noticeably diminished; calcification of the costal cartilages will often be observed.

**Fibrosis.**—This may be unilateral or bilateral; it is shown radiographically as an increase of the radiating striæ, with corresponding loss of translucency and of lighting-up in

## X-RAYS, DIAGNOSTIC USES OF

the affected area. Deformities and limitation of movement of the diaphragm are often seen, and displacement of the heart towards the affected side is common in unilateral examples of this condition.

**Inflammatory lesions.**—1. *Acute lobar pneumonia* produces a fairly dense opacity of uniform distribution, limited to one or more lobes of the lung. Lighting-up is absent in the affected area, but the unaffected portions of the lungs are hypertranslucent. If one lung only is involved, the movements of the diaphragm on that side are diminished or completely arrested. In chronic (unresolved) lobar pneumonia the opacity becomes less complete and its margins are less well-defined. Considerable fibrosis is generally seen, and displacement of the heart towards the affected side is not unusual.

2. In *acute broncho-pneumonia* the radiographic appearances are not at first greatly modified. In the later stages of the disease, however, numerous rounded opacities of some size will be seen scattered throughout the lung substance, together with general diminution of air-entry as recognized by lighting-up on inspiration. Failure of resolution is accompanied by increased fibrosis.

3. *Abscess of the lung*, occurring in the course of an acute pneumonia, is frequently very difficult to recognize by radiographic means, since the opacity of the surrounding consolidated lung generally obliterates all traces of the abscess cavity. Chronic lung abscess is shown as a rounded or irregular area of either increased or diminished translucency, according to the nature of the contents of the cavity (air or fluid) at the time of the examination. Examination in the upright position may show a horizontal fluid level below an area of increased translucency. Fibrosis is always present around a chronic abscess cavity.

**Pulmonary tuberculosis**, in the active state, produces diminished air-entry, limitation of diaphragmatic movement, and, as seen in the radiogram, groups of small rounded opacities with somewhat ill-defined margins. The distribution of these opacities may be apical, perihilar, or diffuse; in the latter case the distinction from broncho-pneumonia caused by other organisms can only be made with difficulty, but the grouping of the opacities and their small size are usually very characteristic.

Contributory evidence is afforded by the phthisinoid shape of the thorax, and the small transverse diameter of the heart.

X-ray examination is of particular value in the perihilar type of tuberculosis, as well marked changes are often observed long before the disease is manifest to other methods of investigation.

In no case of doubtful phthisis, however, can the absence of confirmatory evidence be regarded as definitely excluding the possibility of disease. Advanced cases of pulmonary tuberculosis often present a variety of lesions—large areas of consolidation, cavitation, and fibrosis, in the regions longest affected; and groups of the small opacities mentioned above in the more recently involved areas. In healed tuberculous lesions the diffuse consolidation and small ill-defined opacities are replaced by fibrosis, which sometimes encloses areas of calcification. Air-entry improves, but is rarely restored to the normal; it is frequently impossible to state from X-ray examination alone that the lesion is completely healed. (PLATE 45, Figs. 1, 2.)

**New growths of the lung** usually consist of primary or secondary carcinoma. In either case the lesion produces, on radiographic examination, a number of discrete opacities, larger, denser, and much more clearly defined than the opacities of phthisis. The opacities are generally seen to be widely scattered throughout the lung substance, and are not accompanied by fibrosis. There is frequently considerable enlargement of the bronchial and aortic glands.

**Hydatid cysts of the lung** are seen as rounded areas of uniform opacity, with perfectly regular and clear-cut margins. There are no changes in the surrounding lung tissue. (PLATE 45, Fig. 3.)

**Pleurisy.**—In affections of the pleura, fluoroscopy forms a valuable method of investigation. In *acute* pleurisy without effusion the movements of the diaphragm on the affected side are diminished or completely arrested, but no other abnormality will be observed. In *chronic* pleurisy without effusion the thickened pleura produces a shadow at the base of the thorax which must be differentiated (1) from *pleural effusion*, (2) from *consolidation of the lung*. As regards (1), the margins of the thickened pleura are less well defined and the diaphragm less completely obscured than in the case of effusion; the opacity produced by a thickened pleura is also generally less dense than that of effusion. As regards (2), the thickened pleura does not completely obscure the lighting-up of the underlying lung on

## X-RAYS, DIAGNOSTIC USES OF

inspiration; the shape of the opacity is also of some value in forming an opinion.

**Pleural effusion**, whether serous or purulent, produces a dense uniform opacity, somewhat triangular in shape. The base of the triangle corresponds with the diaphragm, and completely obscures that structure. The inner border of the opacity is concave internally, and the upper part of this border tends to be nearly vertical in direction.

In large effusions the lung on the affected side shows increased opacity owing to partial collapse, and the heart is frequently displaced towards the sound side. If the effusion is not localized, the upper limit is seen to vary with the position of the patient.

**Pneumothorax**.—This is readily recognized by the greatly increased translucency of the affected side of the chest, and the complete absence of the radiographic appearances characteristic of lung tissue. The collapsed lung can be seen as a fairly dense opacity in the costal groove, the heart and great vessels being displaced towards the unaffected side.

In **hydro-pneumothorax** and in **pyo-pneumothorax** the appearances described above are modified by the dense opacity of the fluid. This always presents a horizontal upper border when the patient is erect, and this horizontal border is maintained when the patient is made to bend from side to side, while vigorous shaking movements will produce visible ripples and splashing on the surface of the fluid.

### V. ALIMENTARY SYSTEM (including Accessory Organs)

#### 1. THE TEETH

Abnormalities in the number, shape, and position of the teeth can be recognized without difficulty. (PLATE 45, Fig. 4.) Roots of teeth remaining after incomplete extractions are as a rule readily seen, becoming more opaque, as in the case of sequestrum formation; but when longstanding inflammatory changes are present the fragment of tooth remaining may show a faint opacity owing to absorption of its calcium salts.

Dental caries is shown in radiogram as a rounded translucent area, with well-defined margins, in the crown of the tooth. The outline of the tooth may appear normal in the radiogram, and small cavities involving the inner and outer wall of the crown may thus be overlooked unless great care be exercised. It must also be noted that while most of the

dental fillings in common use are densely opaque to X-radiation, some of them are translucent.

Inflammatory conditions involving the periodontal membrane and surrounding alveolus result in radiographic appearances which are more distinct in the bone than in the tooth, and are identical with inflammatory lesions of bone in other situations—i.e. rarefaction, absorption, necrosis, abscess-formation, and sequestrum-formation. In chronic periodontitis, however, small exostoses are frequently seen projecting from the tooth at the junction of the neck with the crown, and in chronic apical abscess absorption of the apex of the tooth is sometimes present.

Dental cysts present the same radiographic appearances as other cysts of bone, as do also odontomes. The follicular odontome can generally be recognized by the presence of an imperfectly formed tooth in some part of the cyst-cavity, while the epithelial odontome may be seen to be multilocular. Dental cysts and odontomes connected with the lower molar teeth often involve the ascending ramus of the mandible.

#### 2. THE SALIVARY GLANDS

Salivary calculi are most common in the submaxillary gland and its duct, and in the latter situation can be best shown by means of a large dental film placed in the floor of the mouth. Salivary calculi show a somewhat uneven opacity, the outline of which is irregular if the calculus has been formed in the gland, but oval and regular if the duct is the site of formation.

#### 3. THE PHARYNX AND ŒSOPHAGUS

Apart from the detection of opaque foreign bodies in the pharynx or œsophagus, the X-ray investigation of these structures is carried out by observing on the screen the passage of an opaque substance swallowed by the patient. Emulsions of pure barium sulphate or bismuth carbonate are commonly employed. It is of importance that the passage both of the fluid emulsion and of a semi-solid bolus should be observed; for the latter purpose small pieces of bread soaked in the emulsion serve admirably.

The pharynx and the cervical portion of the œsophagus are best examined with the patient erect in the lateral position, while the thoracic portion of the œsophagus is best seen with the patient erect in the right lateral oblique position, except in the lower two or three inches

## X-RAYS, DIAGNOSTIC USES OF

of its course, for which the left lateral oblique position is more advantageous.

In a normal subject, fluid is seen to pass very rapidly through the pharynx and œsophagus as far as the cardiac orifice of the stomach, above which the fluid can be seen to collect for a few seconds. The cardiac sphincter normally relaxes immediately the first portion of the fluid reaches it, and the short delay noted is due to the narrow lumen of the cardiac orifice as compared with that of the œsophagus.

When a semi-solid bolus is swallowed, the passage through the œsophagus is slower, peristalsis of the œsophagus is observed, the bolus is elongated, and a hesitation in the downward progress is frequently observed at the level of the aortic arch.

**Pharyngeal or œsophageal obstruction** from any cause produces a marked delay in the passage of the semi-solid bolus, and, if the obstruction is considerable, in the passage of the liquid also. If the obstruction affects the pharynx or upper end of the œsophagus, violent efforts at deglutition are observed, and the bolus is frequently ejected. Obstruction in the middle and lower portions of the œsophagus is generally associated with vigorous peristalsis, and also reverse peristalsis, of the gullet above the blockage; there is sometimes marked œsophageal dilatation, below which a thin irregular stream of opaque material may be observed passing through the constriction. (PLATE 45, Fig. 5.)

Pharyngeal and œsophageal obstruction may result from intrinsic lesions (e.g. fibrous stricture, neoplasm, etc.), or from pressure on the outer surface of the tube (e.g. aneurysm, retropharyngeal and dorsal abscesses, masses of enlarged glands in the posterior mediastinum, etc.). The first of these groups is usually associated with some degree of muscular spasm; while at the lower end of the œsophagus a purely spasmodic obstruction is sometimes met with—œsophagectasia—the causation of which is unknown.

The precise nature of the lesion causing pharyngeal or œsophageal obstruction cannot be stated from X-ray examination, but intrinsic strictures can generally be differentiated from obstruction due to external pressure, by the demonstration in the latter instance of the primary morbid condition (e.g. aneurysm, spinal caries, etc.). Œsophagectasia can only be diagnosed from organic obstruction by repeated examinations, during one of which

total relaxation of the spasm may be observed. The obstruction in this condition is situated immediately above the cardiac orifice of the stomach. (PLATE 45, Fig. 6.)

Obstruction at the cardiac orifice of the stomach may closely simulate œsophageal stricture, but in this connexion it is of importance to remember that the cardia lies opposite the eleventh dorsal vertebra, and that the lower end of the œsophagus is therefore overshadowed by the diaphragm for a considerable extent in the right lateral oblique position of the patient. This overshadowing is not seen in the left oblique position.

**Pharyngeal pouches or diverticula** arise at the junction of the pharynx with the œsophagus, and when large and distended with food may cause obstruction by pressure on the gullet. They may fill with opaque material, and then present a persistent pear-shaped shadow lying to one side of or behind the upper end of the œsophagus.

### 4. THE STOMACH

The gastro-intestinal tract is investigated by means of the *opaque meal*, and, in the case of the large bowel, the opaque enema also. The essential points as regards the technique of the opaque meal are:

- (1) That the meal should be palatable.
- (2) That it should replace one of the meals usually taken (preferably breakfast).
- (3) That no aperient should have been taken for twenty-four hours prior to the examination.

The quantity of the opaque salt, and the choice of the salt used, are of little importance provided that the operator is thoroughly familiar with the X-ray appearances in the particular circumstances which he imposes.

The following description, except where otherwise stated, applies to a meal composed of  $\frac{3}{4}$  pint of milk, 1 oz. of bread, and sugar to taste, to which has been added 2 oz. of bismuth carbonate.

**Filling of the stomach.**—This should be observed on the screen with the patient in the upright position. A small quantity of the meal at first collects below the *magenblase* (the bubble of gas in the fundus of the stomach), and then gradually canaliculizes the stomach as far as the pylorus. If the stomach already contains fluid the opaque meal is seen to sink through this at several places, and in this case also the *magenblase* presents a straight lower margin



Fig. 1.—Miliary tuberculosis; abscess in right lung.



Fig. 2.—Diffuse pulmonary tuberculosis.



Fig. 3.—Hydatid cyst of right lung.



Fig. 4.—Impacted lower wisdom tooth.

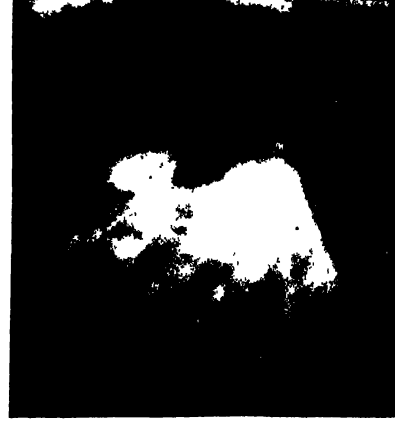


Fig. 5.—Esophageal stricture.



Fig. 6.—Esophagectasia.





Fig. 3.—Ptosis and hypotonus of stomach.



Fig. 2.—Hour-glass stomach.



Fig. 1.—Normal stomach and duodenum



Fig. 6.—Carcinoma of stomach (gastro-jejunostomy has been performed).



Fig. 5.—Penetrating ulcer on lesser curvature of stomach.



Fig. 4.—Atonic stomach.

## X-RAYS, DIAGNOSTIC USES OF

instead of being nearly spherical as in the empty stomach.

When the meal has all been taken the stomach should be examined with reference to shape, position, tonicity, motility, and opening of the pylorus.

(1) **Shape.**—Two types of normal stomach can be recognized, the vertical and the horizontal type. The *vertical* type of stomach is shaped like the letter J. The long limb of the J is cylindrical in form, while the short limb narrows gradually to the pyloric sphincter, at which point the stomach has a blunt, rounded outline.

The *horizontal* type of stomach differs from the above description in resembling a cow's horn in shape rather than the letter J.

The stomach may be deformed as a result of spasm of a group of muscular fibres. The most common site for this spasm is in the more vertical portion of the viscus, where it produces the well-known *spasmodic hour-glass constriction*. Spasmodic deformity may be purely functional; or associated with lesions of other organs, such as phthisis, chlorosis, etc.; or the spasm may be excited by some gross intrinsic lesion of the stomach, such as ulceration. Spasmodic hour-glass constriction always deforms the greater curvature more than the lesser (PLATE 46, Fig. 5).

Deformities of the stomach may also arise from contraction of scar tissue (*organic hour-glass constriction*), from projection into the lumen of growths of the stomach-wall, and from pressure of adhesions and tumours external to the stomach. (PLATE 46, Fig. 2)

(2) **Position.**—The normal stomach lies much lower in the abdomen, and more nearly vertical, than anatomists have supposed. The organ slopes downwards and forwards from the cardia at an angle of about 40° with the vertical; the lowest point on the greater curvature of the empty stomach lies at the level of the umbilicus, or very little higher. When the organ is full the greater curvature commonly reaches a point 1½–2½ in. below the umbilicus. If the latter figure is exceeded some degree of *ptosis* is present (PLATE 46, Fig. 3).

(3) **Tonicity.**—The tonicity of the stomach is normally responsible for supporting a large quantity of the contained meal in the form of a vertical cylinder (*orthotonic stomach*, PLATE 46, Fig. 1). Should the tonicity be defective, the meal tends to accumulate in the lowest part of the organ. Mild degrees of this defect are

described as *hypotonus*, severe degrees as *atonia* (PLATE 46, Fig. 4). If the tonicity is greater than normal (*hyperlonus*), the lower part of the stomach is drawn up, so that the greater curvature comes to lie at a much higher level, as compared with the pylorus, than is the case in the orthotonic stomach.

(4) **Motility.**—The motility of the stomach takes the form of a peristaltic wave, starting just above the middle of the greater curvature. It is seen as an indentation of the greater curve, with a corresponding smaller indentation of the lesser curve at a point opposite. The peristaltic wave passes down towards the pylorus, becoming deeper as it progresses, until finally, at a point 1–1½ in. from the pylorus, the indentations on the greater and lesser curves practically meet, cutting off the pyloric antrum from the remainder of the viscus. The wave passes right down to the pylorus, where it gradually disappears. Peristalsis is normally visible as soon as the meal enters the stomach, and continues until the organ is empty. Each wave should take 15–20 seconds to complete its course down the organ, and the succeeding wave should be visible by the time the first has disappeared. (PLATE 46, Fig. 1.)

In *hypomotility* the indentations of the gastric outline are more shallow, start at a lower level, proceed more slowly, and succeed each other at longer intervals. They may disappear entirely before reaching the pylorus.

In *hypermotility* the indentations are seen at an unusually high level, are very deep, and progress rapidly; while three, four, or even more distinct peristaltic waves may be present at the same time.

(5) **Opening of the pylorus.**—The time which elapses between the taking of the meal and the first opening of the pylorus depends, in a normal subject, on the consistency of the food in the pyloric antrum. When the meal specified above is given, the pylorus should open in from five to ten minutes, the latter being the maximum time in normal persons. (If an opaque drink is given, this starts to leave the stomach immediately.) The relaxation of the pyloric sphincter occurs when a peristaltic wave has approached to within ½–1 in. of it. A very thin stream of opaque chyme is seen to pass through the pyloric canal, and this immediately spreads out in the duodenum to form the "duodenal cap" (see p. 558). The entrance of the chyme into the duodenum is quickly followed by closure of the pylorus (pyloric reflex). Once the pylorus has begun to open, it

## X-RAYS, DIAGNOSTIC USES OF

continues to do so at fairly regular intervals until the stomach is empty, but not necessarily in response to every peristaltic wave.

*Incompetence* of the pylorus is sometimes seen when the pyloric region of the stomach is infiltrated with malignant growth.

*Pyloric stenosis* may be spasmodic or organic, or the two conditions may be combined. The radiographic diagnosis of organic pyloric stenosis depends upon the recognition of persistent failure of the normal sphincteric relaxation, often combined with some deformity of the pyloric end of the stomach. In recent cases the stomach is ortho- or hyper-tonic, and the motility is exaggerated, in spite of which there is more or less delay in the emptying of the organ. Wide, purposeless movements are frequently observed in the pyloric antrum, and a characteristic dilatation of the antrum towards the right (prognathous antrum) often occurs.

Later, the stomach becomes atonic, and the motility defective or even absent for long periods, and retention of the meal may be greatly prolonged.

Spasm of the pylorus may be purely functional, or result from hyperchlorhydria, pyloric ulcer, or delay in the passage of the meal through any part of the small gut.

*Emptying-time of the stomach.*—The average time in which the meal should have left the stomach is four hours. Undue rapidity of emptying is occasionally seen in duodenal ulcer, or may be purely functional. Delay in emptying may result from defect in any of the five functions discussed above.

**Gastric ulcer.**—The X-ray diagnosis may be direct or indirect. *Direct* diagnosis depends on observation of the filling of the crater of the ulcer with the opaque meal. This produces a localized projection from the stomach outline; very occasionally a small bubble of gas is seen in the deepest part of the ulcer above that part filled by the meal. (PLATE 46, Fig. 5.) Direct diagnosis of an ulcer can be made with absolute certainty, but it is impossible to state whether the ulcer is benign or malignant.

*Indirect* diagnosis of gastric ulcer radiographically rests upon a combination of several of the following phenomena :—

- (1) Delay in emptying of the stomach.
- (2) Local deformities due to spasmodic or cicatricial contraction, or adhesion to other structures.
- (3) Persistent defect of "jump-gap" in the peristaltic wave at one spot.

Indirect diagnosis can rarely be more than tentative.

Of the deformities which may result from ulcer, the most striking is the hour-glass constriction (described above). *Spasmodic hour-glass constriction* may, however, be present in very many conditions besides gastric ulcer, and an attempt must therefore be made to differentiate this variety from the constriction which is, at any rate in part, organic. The only certain means of ascertaining the spasmodic nature of the deformity is to obtain complete relaxation of the spasm.

This may occur spontaneously, or may be induced by—

- (1) Massage of the abdomen.
- (2) Administration of sodium bicarbonate.
- (3) Administration of atropine. (This, to be effectual, must be pushed to the limits of tolerance.)

**Carcinoma of the stomach.**—An early carcinomatous ulcer cannot be differentiated by X-ray examination from simple ulceration. When carcinomatous infiltration of the stomach-wall has occurred to any considerable extent, however, the X-ray appearances are generally typical, and consist of marked defects in the outline of the organ, with absence of movement in the affected portion (PLATE 46, Fig. 6). This "filling-defect" is best seen when the body of the stomach is involved; carcinoma of the pylorus generally presents the features of an early pyloric stenosis, but occasionally the pyloric sphincter is incompetent.

The form of scirrhus carcinoma which causes the *leather-bottle stomach* produces very characteristic evidence: the stomach appears extremely irregular and constricted throughout its entire extent; it is absolutely inert, and the meal passes with great rapidity into the duodenum, as a consequence of the incompetence of the pylorus.

### 5. THE SMALL INTESTINE

The chyme which enters the *duodenum* at each relaxation of the pyloric sphincter spreads out in the first  $\frac{1}{2}$ – $\frac{3}{4}$  in. of the gut and produces a dense regular shadow known as the "duodenal cap." This shadow is generally cone-shaped, the base of the cone resting upon the flat duodenal face of the pyloric sphincter, and commonly persists unaltered for an appreciable time. The chyme forming it is then passed through the remainder of the duodenum in a much more finely divided form, and with some rapidity. It is commonly arrested temporarily

## X-RAYS, DIAGNOSTIC USES OF

at two points—(1) just below the entry of the common bile-duct into the gut, and (2) half-way along the transverse part of the duodenum. Transitory retrograde movement of the chyme is observed to originate from both these points. Delay in the passage of the chyme at the duodeno-jejunal flexure does not occur except as a result of gross obstruction.

Elongation and dilatation of the first part of the duodenum is observed in **gastroptosis**; otherwise dilatation of the duodenum is only seen in the presence of obstruction to its lumen, e.g. pressure of adhesions or of an enlarged pancreas.

**Duodenal ulcer** occurring in the first part produces persistent deformity of the duodenal cap. The crater of the ulcer is sometimes filled, but the deformity is usually in the nature of a defect (filling-defect) in the outline of the cap. (PLATE 47, Fig. 1.) This is combined with gastric hypertonus, exaggerated peristalsis, and free opening of the pylorus when the meal is first taken. These latter phenomena rarely persist, and complete emptying of the stomach is generally delayed; in a certain proportion of cases of duodenal ulcer, however, the entire meal leaves the stomach with great rapidity, even in half an hour or less. Chronic ulceration in this situation not infrequently results in some degree of pyloric stenosis.

Ulceration of the duodenum beyond the first part is very rare; the crater of the ulcer can sometimes be seen filled with opaque chyme.

**Jejunum and ileum.**—Owing to the state of fine division of the chyme in the jejunum and upper two-thirds of the ileum, these portions of the gut can only be satisfactorily studied when large quantities of the opaque salt have been given. At least two forms of intestinal contraction will then be seen—(1) peristaltic waves which affect short segments of the gut, and pass the contents rapidly along for two or three inches or more, before subsiding, and (2) narrow, stationary segmental constrictions, which divide the contents of the affected length of gut. Segmental constriction is often combined with localized antiperistalsis.

The lower third of the ileum, when filled, can always be seen, owing to the greater concentration of the unabsorbable opaque salt in that part of the gut. The intestinal movements are similar to those in the more proximal portions of the small gut, but are generally much less active; if, however, the patient is observed while taking food from four to five

hours after the opaque meal, it will be seen that entry of food into the stomach produces active peristalsis in the lower ileum, relaxation of the ileal sphincter (which probably consists of a specialized band of circular muscle-fibres in the terminal inch of the ileum, providing, by means of tonic contraction, the true sphincter between the large and small gut), and resultant rapid entry of chyme into the caecum. This phenomenon is known as the "gastro-ileal reflex."

The time normally occupied in passage through the jejunum and ileum is about three hours. A further  $\frac{1}{2}$ -1 hour usually elapses after the arrival of the first portion of the chyme in the lower end of the ileum before any relaxation of the ileal sphincter permits of entry into the caecum. Hence it is obvious that opaque material will be present in the lower ileum for at least four hours after the stomach is empty. Food or drink taken before the opaque meal has completely left the stomach will mix to some extent with the remaining opaque salt, and so produce an apparent delay in the passage of the opaque meal through the small gut. The failure to recognize this fact is responsible for many fallacious diagnoses of "ileal delay." The most reliable evidence of true ileal delay is provided by observation of the time of entry of the first portion of the opaque meal into the caecum. If the caecum contains no opaque material at the fifth hour (or later), ileal delay can safely be recorded, always provided that the passage through the stomach and jejunum has taken place normally. True ileal delay may be purely functional, or may result from delay in the proximal portion of the large gut, from inflammatory lesions of any of the abdominal organs (especially those in the pelvis) or from gross obstruction (e.g. adhesions). Ileal delay may produce gastric delay by causing spasm of the pylorus, thus increasing the difficulty of diagnosis.

### 6. THE LARGE INTESTINE

(1) **Position.**—The position of the large gut is seen to vary greatly in apparently normal subjects, and in the same subject with changes of posture. In the upright position it may be stated that the caecum should be contained almost entirely in the iliac fossa, that the hepatic flexure should lie at least  $1\frac{1}{2}$  in. above the iliac crest, and that the splenic flexure should be 3-6 in. higher. The transverse colon normally descends 3-5 in. below the umbilicus.

## X-RAYS, DIAGNOSTIC USES OF

*Ptois* of the large gut, as judged by these standards, is exceedingly common, and is frequently unaccompanied by any symptoms. Ptois is frequently associated with delay in the passage of the contents through the distal portion of the gut; the ptois appears, however, to have little if any influence in producing this delay, and probably merely forms one manifestation of general muscular atony, of which the intestinal delay forms another manifestation. (PLATE 47, Fig. 2.)

(2) **Movements of the large gut.**—The contents of the large gut are not propelled onwards by a peristaltic wave, but by occasional simultaneous contractions of the circular muscle-fibres over a considerable length of gut. The movement of the contents thus produced is known as "mass-movement." These contractions are of short duration, and may occur only two or three times in twenty-four hours. They are nearly always brought about by the entry of food into the stomach (gastro-colic reflex).

In the normal subject the rectum never forms a reservoir for the faeces; the entry of faecal material into this portion of the gut immediately produces a desire for defaecation.

(3) **Rate of passage.**—The rate of passage of the contents through the large bowel presents many anomalies in different normal persons, and in the same person at different times; it must be remembered that this part of the alimentary tract contains, during all or part of the examination, the residue not only of the opaque meal, but also of food taken before and after the opaque meal. It is, in consequence, exceedingly difficult to draw any reliable deductions from variations in the rate of passage through this portion of the gut.

Provided that the bowels act daily before and during the examination, the figures that follow will give some general indication of the period at which the various portions of the gut should be filled. It can, moreover, be stated that delay in the proximal portions of the large gut is exceedingly rare in the absence of organic obstruction. The times given refer to the point reached by the most distal portion of the opaque material. No importance can be attached to the position of the remainder, owing to the nature of the large-gut contractions (see above), and the inevitable mixing, in the caecum and ascending colon, with the residue of meals taken before and after the opaque meal.

- (1) The caecum should start to fill in 3½–4½ hours.
- (2) The hepatic flexure should be reached in 6–8 hours.
- (3) Most of the transverse colon should be filled in 10–18 hours.
- (4) The pelvic colon should contain some of the opaque material at the end of 24 hours.

If the bowels continue to act daily, the whole of the opaque residue will often be evacuated in 72 hours or less, but wide variations occur in perfectly normal subjects.

**Constipation.**—Constipation occurring apart from an organic obstruction may be due (1) to delay in the colon, caused either by atony or by spasm of the bowel musculature, (2) to inefficient defaecation-dyschezia (Hurst). In the latter type no delay is seen in the passage through the colon, but the rectum contains faecal material after defaecation.

### **Organic obstruction of the large gut.**

—This can only be recognized when very prolonged delay occurs at one point, and persists in spite of one or more actions of the bowels. In these circumstances confirmatory evidence should be obtained by administration of an opaque enema (see p. 561). The nature of the obstruction cannot generally be stated from X-ray examination.

It cannot be too strongly emphasized that minor variations in the rate of passage of the contents of the large gut are of no diagnostic significance whatsoever.

**The vermiform appendix.**—Special technique is required to demonstrate this organ. A dose of castor oil should be administered twenty-four hours before the examination, and the meal consist of an emulsion of barium in butter-milk or water. The appendix is usually found to be filled at the eighth or the twenty-fourth hour after this meal. If no view has been obtained by the twenty-fourth hour, an aperient should be given and the meal repeated. By following this technique the appendix can be seen in practically every case when normal, and in a very large proportion of cases when abnormal. The mobility of the appendix, irregularities of lumen, the presence of concretions, and localized tender points, etc., can readily be demonstrated. This is the direct method. (PLATE 47, Figs. 3, 4.) If observation of the appendix cannot be obtained in this way, the indirect method should be adopted. For this purpose the usual meal of bread and milk with the opaque salt is given, and the

## X-RAYS, DIAGNOSTIC USES OF

patient takes his meals at the ordinary intervals throughout the day. Chronic inflammatory changes in the appendix can then be inferred from observation of the following phenomena :

1. Marked ileal delay persisting up to the eighth hour after the opaque meal.

2. Localized tenderness over the ileo-cæcal junction (this can only be recognized by palpation under the fluorescent screen, owing to the wide variations in the position of the cæcum).

3. If extensive adhesions have formed, the terminal ileum and the cæcum may be found to possess greatly diminished mobility.

**Diverticulitis.**—In this condition the diverticula may be seen filled with the opaque material. The diagnosis can only be definite if the supposed diverticula are still visible after the remainder of the opaque material has been evacuated. Confirmation may sometimes be obtained from the administration of an opaque enema.

**The opaque enema.**—This is administered after the large gut has been emptied as far as possible by means of purgatives and enemata. Opaque salt 6–8 oz. is suspended in 2 pints of warm water, to which a small quantity of methylated spirit may be added with advantage. This is introduced slowly through a rectal tube with funnel under direct fluoroscopic observation. In favourable circumstances the enema should fill the whole of the large gut, and should reach the cæcum in about three minutes.

**Dilatation, stenosis, and diverticula** may be demonstrated by the enema, but too much importance should not be attached to apparent obstruction as shown by this means, unless the appearances are confirmatory of those noted in the passage of the opaque meal. In some normal individuals the large gut cannot be completely filled by the enema. On the other hand, complete rapid filling of the bowel is valuable evidence of the absence of any organic obstruction.

**The liver and biliary passages.**—Active inflammatory lesions in and around the liver (e.g. liver abscess, subphrenic abscess) produce diminution or complete arrest of diaphragmatic movement on the right side. The diaphragm may also be displaced upwards or deformed.

Enlargement downwards of the liver can be recognized in all but very obese subjects, in whom the lower margin of the organ may be indistinguishable.

**Biliary calculi.**—These can be demonstrated in a small proportion of cases. In the

gall-bladder the calculi generally produce a typical appearance if seen at all (PLATE 47, Fig. 5), the margins of the calculi being much more opaque than the central portions. Calculi in the cystic and common bile-ducts are often found to produce a much more homogeneous opacity, which may closely simulate that of a renal calculus. The differential diagnosis in such cases depends entirely on the position of the opacity in relation to the bones, the view being secured in the fixed position.

## VI. THE URINARY SYSTEM

In no branch of radiography is correct technique of such vital importance as in the examination of the urinary tract. The patient must be prepared thoroughly by means of laxatives combined with restriction of diet for two days before the examination. Vigorous purgatives, however, are to be avoided. On the day of the examination the patient should abstain from food till after the visit to the radiographer. PLATE 47, Fig. 6, shows a calculus which repeated examination failed to demonstrate until the patient was admitted to hospital and thoroughly prepared. The routine investigation of the urinary tract should always include both kidneys, the ureters, and the bladder, and this can be accomplished by means of two radiograms taken from below the couch. For examination of the upper part of the tract the patient is placed in the prone position with a pad of cotton-wool in the epigastrium, and the tube is centred over the second lumbar spinous process; if a 10-in. by 12-in. plate is used, both kidneys and the upper part of both ureters can be included in the view.

To deal with the lower part of the tract, which is nearer to the anterior than the posterior part of the body, the patient is placed in the supine position, and the tube is centred over the middle of the symphysis pubis. This view includes the lower ureters, the bladder, and the prostatic areas. Since the kidneys move with respiration, the upper view is taken with the breath held in full inspiration. The lower part of the urinary tract is not influenced by the respiratory movements, but compression is advisable to overcome the movement of the abdominal wall, which would otherwise be communicated to the plate. The view of the upper part of the tract, to be satisfactory and efficient, should show—

1. The margins of the psoas muscles.
2. Clear definition of the lumbar vertebrae and their transverse processes.

## X-RAYS, DIAGNOSTIC USES OF

3. The 11th and 12th ribs.
4. The outlines of the kidneys.
5. The posterior part of the iliac crests.

If the technique described above is observed, the positions of the pelves of the kidneys and of the ureters are as shown in PLATE 48, Fig. 1. Opacities are commonly seen in views of the abdomen and pelvis, which can only be differentiated from those due to urinary calculi by consideration of their position in relation to the bones of the parts. The position of the urinary tract must therefore be a matter of certain knowledge, and not merely of conjecture.

**Urinary calculi.**—With the exception of the pure uric-acid calculus, which for practical purposes may be considered as translucent to X-radiation, all urinary calculi produce a recognizable opacity in a radiogram of good quality. The density of the opacity varies within very wide limits, according to the composition and bulk of the concretion and the obesity of the patient. The opacity is generally of uniform density throughout its extent; sometimes the density is greatest at the centre of the shadow and fades off towards the margins, but this variation is gradual and regular. Occasionally large vesical calculi produce definitely laminated shadows.

No definite rules can be laid down for the shape of urinary calculi, but certain forms are very common and characteristic, e.g. triangular, dumbbell-shaped, and branched shadows in the region of the renal pelvis (PLATE 48, Figs. 2, 3); rounded and fusiform opacities in the line of the ureter (PLATE 48, Figs. 4, 5); round and oval opacities in the bladder area. The position of the opacity in relation to that occupied by the urinary organs is of prime importance in this connexion, but the possibility of anatomical abnormalities must not be forgotten (e.g. double ureter, double renal pelvis). In cases of difficulty, the passage of an opaque ureteric catheter, or the injection of an opaque salt into the renal pelvis, may afford valuable assistance. (PLATE 47, Fig. 5.)

**Differential diagnosis.**—The opacities of urinary calculi must be differentiated from those of—

- (1) Caseous material in the kidney.
- (2) Intestinal contents.
- (3) Calcareous abdominal glands.
- (4) Biliary and pancreatic calculi.
- (5) Calcified concretions in the vermiform appendix.
- (6) Phleboliths.

(1) Caseous material in the kidney produces an ill-defined diffuse opacity, which generally shows irregular variations in density.

(2) Intestinal contents must be eliminated in cases of doubt by further preparation of the patient and re-examination.

(3) Calcareous abdominal glands may closely simulate urinary calculi. Calcareous abdominal glands are, however, (a) generally multiple; (b) often mobile, so that a second examination may show that the suspected opacity no longer lies in the region of the urinary organs. Further, the opacity of calcareous glands is usually very irregular in outline and density, and often definitely punctate. The external iliac glands are frequently calcified, but the opacities produced by these lie external to the line of the ureter.

(4) The diagnosis of biliary calculi has been discussed above. Pancreatic calculi are rarely seen; they lie internal to the pelves of the kidneys.

(5) Calcified appendicular concretions are usually fusiform, but the long axis of the concretion rarely lies in the line of the ureter. The presence of calcified ileo-colic glands may give assistance in pointing to the appendicular nature of the lesion.

(6) Phleboliths are found occasionally in the prostatic and vesical veins. They are very small, often multiple, and some of the opacities will generally lie outside the area occupied by the urinary organs.

In conclusion, it may be stated that clinical confirmation should be sought for in all cases where an opacity suggestive of urinary calculus is present.

**Urethral and prostatic calculi.**—Calculi in the prostatic portion of the urethra produce opacities which lie in the mid-line opposite the symphysis pubis.

Prostatic calculi are usually multiple; they are seen lying just above or overshadowed by the bodies of the pubic bones.

**Tuberculosis of the kidney.**—This condition cannot be diagnosed with certainty by X-ray examination. It may, however, be suggested if one or both kidneys are seen to be greatly enlarged in the absence of calculus, and especially if opacities indicative of calcified caseous material are present.

**Vesical diverticula** are readily demonstrated after injection of the bladder with an emulsion of barium sulphate (PLATE 48, Fig. 6).

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Fig. 1.—Duodenal ulcer.



Fig. 2.—Ptosis of large intestine.



Fig. 3.—Normal appendix.



Fig. 4—Chronic inflammation of appendix.

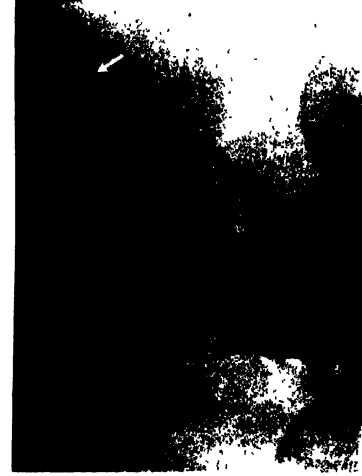


Fig. 5.—Normal pyelogram; a biliary calculus lies external to the renal pelvis.



Fig. 6.—Oxalate calculus in kidney.





Fig. 1.—Shadow relationship of ureters and kidneys. A and B, renal pelvises. Straight lines indicate lines of ureters.



Fig. 2.—Pyuria from calculus in both kidneys.

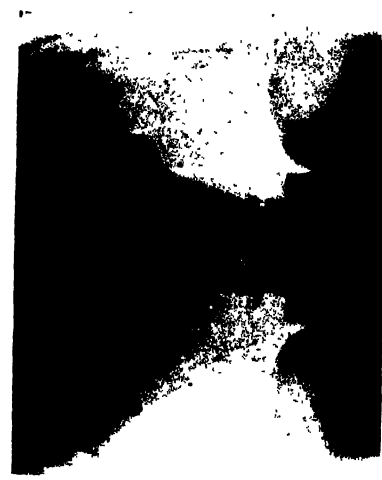


Fig. 3.—Renal calculus.



Fig. 4.—Ureteric calculus.



Fig. 5.—Calculi in lower part of both ureters.

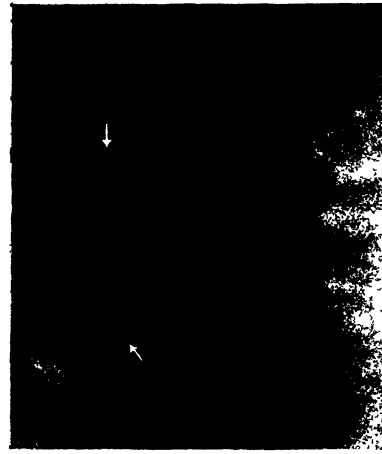


Fig. 6.—Vesical diverticula.

## X-RAYS, THERAPEUTIC USES OF

### X-RAYS, THERAPEUTIC USES OF.

—The X-rays have been found of value in the treatment of a large number of pathological conditions, the chief of which may be grouped thus:—

1. Various diseases of the skin and its appendages, and pruritus.
2. Superficial and deep-seated malignant growths, and the pain and ulceration associated with them.
3. Certain disorders of the spleen, thyroid, and lymphatic glands.
4. Some affections of the genital organs.

It is not necessary to specify every disease which has received benefit from X-ray treatment, nor is it always possible to state exactly to what particular action or variety of the rays the result is due. It is probable that there is a definite effect upon the circulating blood, and that hard rays have a greater therapeutic effect than soft rays. Speaking generally, the properties they possess are made use of for the following purposes:—

1. *Epilation*.—In ringworm and favus of the scalp, ringworm and staphylococcic sycosis of the beard, and hypertrichosis. (On account of the danger of telangiectasis and atrophy of the skin after X-ray treatment for hypertrichosis, electrolysis is the method of choice.)

2. *Inhibitory and atrophic action on glandular tissues*, e.g. sweat- and sebaceous glands in hyperidrosis, acne, etc., thyroid and lymphatic glands and spleen in Graves's disease, lymphadenoma, leukaemia, etc.

3. *Anæsthetic and analgesic action*.—In localized pruritus and pain, e.g. pruritus ani, neuritis.

4. *Depression of activity and destruction of newly formed and actively growing cells*.—In rodent ulcer, carcinoma, sarcoma, mycosis fungoides, and possibly for the same reason in psoriasis, eczema, etc.

5. *Stimulation of growth of healthy tissues and removal of inflammatory deposits*.—In lupus vulgaris, lichenification, leucoplakia, chronic ulcers, fissures, sinuses, etc.

6. *Resolvent or atrophic action on horny, fibrous, and other growths*.—In fibro-myoma of the uterus, prostatic hypertrophy, cheloid, callosities, warts, etc.

**Superficial diseases.**—It will be noticed that most of the affections mentioned above as suitable for or amenable to X-ray treatment are superficial, or directly accessible to the rays. Since these exert their maximum effect on the surface, and also possess a selective affinity for epithelial cells, it is not surprising that their

therapeutic action is turned to account in a large number of cutaneous diseases. Indeed, it might almost be said that nearly all chronic dermatoses derive benefit in greater or less measure from suitable doses of the rays. But success is conditioned not only by the superficial position and chronicity of the lesion: the type of cell, and pathological nature of the lesion, the extent and accessibility of the disease, the tendency of the complaint to relapse, the danger of repeated large doses of the rays, the suitability of other methods, and other considerations must be taken into account before X-ray treatment is adopted.

**Deep-seated diseases.**—The treatment of deep-seated disease presents a somewhat different problem on account of the difference in the technique and because the cases are often beyond the reach of surgery or are not amenable to medical treatment. In treating growths or diseased organs lying beneath the skin, the skin itself must be protected so that the larger quantities of rays required shall not injure it in their passage. This is effected by intercepting the rays of shorter wave-length by means of a filter of aluminium or other material and allowing only the hard gamma radiations to penetrate. Thin aluminium filters may also be employed in treating some skin diseases in which there is much infiltration, e.g. lupus vulgaris. In some cases preparations of silver have been injected into tumours in order to intercept the rays and utilize the secondary rays emitted from the metal particles. Using a filter of 3 mm. of aluminium and employing "cross-fire"—i.e. radiating the tumour from several different directions so that no area of skin is exposed more than once at a sitting—massive doses may be administered to deep-seated malignant tumours, fibroids of the uterus, etc., with resulting diminution or complete disappearance of the growth. Operable malignant growths should be removed surgically, and X-ray treatment reserved for the relief of cases in which this is impossible, particularly with the view of mitigating pain and lessening discharge. Prophylactic raying of the tissues after operation has been adopted by some operators, and a certain measure of success in the prevention of recurrence has been claimed.

**Technique and dosage.**—In general terms it may be said that small doses of X-rays stimulate and large doses depress the activity of the cells or cause their destruction. In order to administer the rays in suitable doses without injuring the skin, some means of measuring

## YAWS

the quality and quantity of the rays is of the first importance. The *quality*—i.e. whether the rays emitted by the tube are soft or hard—can be estimated by means of an instrument such as that of Benoist, consisting of varying thicknesses of aluminium which, when placed in front of the screen, gives the degree of penetration as compared with a central disc of silver; or by means of Bauer's Qualimeter, attached to the cathode of the tube. If the tube is too hard the vacuum can be lowered by a regulating device. Owing to the uniformity of its output and ease of regulation, the Coolidge tube has found much favour both in radiography and in therapeutic work. The *quantity* of rays emitted by the tube is measured by an indicator such as that of Sabouraud and Noiré, which consists of a small cardboard disc or "pastille" coated with platino-cyanide

of barium, placed midway between the anti-cathode of the tube and the skin, and which changes from a green to a standard brown tint after it has been exposed to the rays for a certain time. The brown tint, "teinte B," represents the maximum dose which can be administered with safety in one application when no filter is used, and is also the dose required to produce epilation. An exposure of this strength may be repeated at intervals of three weeks or a month, and is a suitable dose for many skin diseases. Fractional doses can be measured by Sabouraud's pastilles and Corbett's tintometer, and may be administered at shorter intervals. For the deeper structures a filter may be used, and several pastille doses may then be given at a sitting, according to the thickness of the filter employed.

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**YAWS** (*syn.* *Framboesia*, Boubas (Brazil), Coko (Fiji), Parangi (Ceylon), Dube (Gold Coast).)—A contagious disease of hot countries caused by the *Spironema pertenu*, and resembling syphilis in many of its manifestations.

**Geographical distribution.**—The disease has a wide distribution in the tropics, especially in West Africa, though it is found throughout the central part of that continent; it occurs in many of the West India Islands, in Ceylon, in Fiji, Samoa, and many other islands of the Pacific, also in Papua, Java, Sumatra, and the Malay States. The exact distribution in India and China is unknown. In Fiji it would seem that every child passes through an attack before reaching maturity. Owing to its great similarity to syphilis, and the extent to which the two diseases overlap, it would appear to be impossible to give its exact range at the present day. Wherever yaws is widespread—as, for instance, in Fiji—syphilis appears to be unknown among the native tribes.

**Etiology.**—The *Spironema pertenu*, discovered by Castellani in 1905, is a fine thread-like corkscrew organism consisting generally of 8–12 convolutions, but sometimes of as many as 20; it measures up to 20  $\mu$  in length. The method of infection is *extragenital*, by direct contact through abrasion of the skin,

and therefore the disease is most widespread in closely crowded native communities.

**Pathology.**—There are no gross changes peculiar to yaws, but in the microscopic pathology the absence of the endarteritis which occurs in syphilis is to be noted.

**Symptomatology.**—The incubation period appears to be two to three weeks in man; experimentally in apes it may be as long as three months. The **primary lesion** appears as an isolated papule, or it may be a bulla, which develops a few days later into an ulcer with a raw base and undermined edges. Accompanying it are headaches, bone-pains, pyrexia, and gastric disturbances—evidences of a systemic intoxication; in children there may be diarrhoea. The primary lesion may be so small as to be overlooked, and great difficulty may be experienced in distinguishing it from many cutaneous lesions so common in the tropics. While always extragenital, it may be situated on any part of the legs or trunk.

The **secondary stage** is characterized by more pronounced symptoms of toxæmia and a peculiar eruption, which breaks out after an interval of a few weeks up to three months from the primary lesion. Typically, it consists of strawberry-coloured papillomata, though, as in syphilis, the eruption is pleomorphic; sometimes it appears as a roseola, at others as papules

## YAWS

or macules accompanied by desquamation resembling a squamous syphilide. This furfuraceous desquamation, when slight, may easily be overlooked; when pronounced, oval or circular rings appear surrounding areas of healthy skin. After a variable period, papules are seen in the furfuraceous patches, commencing around hair-follicles, and exuding a tenacious cheesy substance which can only be

individual yaw may be no larger than a pea, or may attain the size of a half-crown. It is tipped with a yellowish-grey crust standing out  $\frac{1}{4}$  in. from the healthy skin. It becomes dark brown as if ingrained with dirt, even black in colour, though appearing light on a black skin, owing to destruction of the pigment layer; in white races it is coppery-red. When the crust is removed, bleeding granula-



Fig. 109.—Tibial periosteal nodes, ulcers, and deformity of phalanges. (*Manson-Buhr.*)

removed with difficulty. They occur in groups, a larger one appearing to give off a series of smaller satellites. Auto-inoculation is probably responsible for the symmetrical appearance of these lesions wherever the skin or mucous surfaces come in contact. Thus they are present at the angles of the mouth, in the axilla, round the anal cleft, and between the thigh and the scrotum. In contradistinction to syphilis, they are rarely found on the true mucous surfaces, but often occur in clusters just inside the nostril. Several of these groups of yaws may coalesce so as to cover a large surface, as the entire cheek or the popliteal space. The

tissue remains, unless secondary infection causes pus-formation. The itching and irritation may be very considerable. Simultaneously with the appearance of the eruption, as in syphilis, a uniform symmetrical, painless enlargement of the lymphatic glands may take place, and in the lymph aspirated from them spirochetes have been demonstrated. With this exudate Nicholls has succeeded in infecting rabbits by intratesticular injection, and Castelli by the intravenous route reproduced secondary lesions in these animals. Small yaws may form in and around a nail, giving rise to a particularly intractable onychia. When the

## YAWS.

yaws develop on the sole of the foot, they give rise to widespread ulceration, attain a large size, and cause a considerable amount of pain. They begin as tumours beneath the epidermis, and, being under tension, are exquisitely tender; when the tough epidermis gives way they leave behind radiating cracks.

After the secondary stage has lasted two or three months, absorption of the papillomata takes place, generally beginning in the oldest of the lesions and leaving the margins of the yaws still active, thus causing a ringworm-like appearance.

**Tertiary stage.**—The lesions ascribed to the tertiary period are manifold. Generally speaking, they consist of extensive ulcerations occurring at the site of the secondary yaw. They may be of large size and may be situated on any part of the body, but are more usually found over the external malleolus and on the extensor aspect of the leg. They may persist for months or years, recurring from time to time as fresh outbreaks.

Periosteal bone-pains, becoming more intense at night, especially in the long bones, are a persistent and distressing sequela. They are probably due to a rarefying osteitis, which is succeeded by acute periostitis, periosteal nodes, and sabre-shaped bending of tibiae and forearms. Epiphysitis, deformity of the hands and shortening of the digits, and a general diffuse periosteal thickening, usually of the clavicle, have been noted. (Fig. 109.)

Rarefying osteitis, when it attacks the long bones, may lead to spontaneous fracture and consequent malunion. A tertiary synovitis of the knee-joint, which may produce complete disorganization, has been observed.

Gumma-like lumps may form and eventually ulcerate in almost any part of the body, especially over the sternum and on the legs; in the latter situation such extensive cicatrization may ensue as to lead to a chronic lymph-stasis and an elephantoid state of the limb.

In Fiji tertiary ulcerations of the lower lip are frequently seen, leading eventually to macrocheilia. Finally, ulceration of the nasal septum, which proceeds to total destruction of the nose, may occur. This unsightly condition, which is regarded by some as being but a tertiary manifestation, is known as *gangosa*, and is very common in certain of the West Indies, Fiji, Papua, and British Guiana. The ulceration may involve the palate and larynx.

Yaws appears to be neither hereditary nor

congenital, for a child born of a yaw-stricken mother is healthy, and only contracts the disease through a breach of surface while suckling. Pregnant women stricken with yaws are likely to abort.

Lately, in Fiji, Harper has seen general-paralytic cases, tabetics, and other paraplegias which, after full investigation, can only be ascribed to this disease. The Wassermann reaction in yaws is positive as in syphilis, and is most pronounced during the secondary eruption.

**Diagnosis.**—Yaws has to be distinguished, especially in its later stages, from *syphilis*. This is by no means always an easy matter; Hutchinson, indeed, believed the former to be merely a tropical variety of the latter. The differences between the two diseases have already been emphasized.

There appears to be a reciprocal immunity between them. Monkeys inoculated with yaws possess a certain degree of immunity against syphilis. Both diseases may, however, occur in the same individual. The ulceration of the nose and face roughly resembles *leprosy*, but the formation of anæsthetic spots and the discovery of the acid-fast bacillus in that disease should render the diagnosis a matter of no very great difficulty. *Gangosa* must be distinguished from *naso-pharyngeal leishmaniasis* ("bubas Braziliana" or "uta"), diseases which generally involve the soft palate, larynx, and nasal septum: this is easily done in practice by demonstration of the Leishman-Donovan body in the ulcerations. From nasal *lupus* it has to be differentiated by the slow progress, involvement of surrounding skin, and escape of bony tissue from destruction in this disease.

The secondary furfuraceous rash has to be distinguished from the dermatomycoses, *Tinea albigena* and *T. circinata*. Ulcerations of the leg may be mistaken for the common *ulcus tropicum* or a skin blastomycosis.

**Prognosis.**—Although the mortality directly ascribable to yaws is not great, the disease causes considerable physical disability, especially in the tertiary stages.

**Treatment.**—Salvarsan or, better still, neo-salvarsan, galyol, kharsivan, and arsenobillon, all exert a remarkably rapid curative effect. In Ceylon, injection of 3 to 5 c.c. standard solution of mercury or arsenious iodide has been followed by good results, but salvarsan acts more rapidly. The more urgent symptoms yield much more readily than in syphilis, and relapses are uncommon. The salvarsan

## YELLOW FEVER

compounds should be given by the intravenous route in doses of 0.6 grm. for three injections at intervals of a month. Strong believes in smaller doses, 0.3 grm., repeated every fortnight. Doses smaller than in syphilis are sufficient; some believe that the majority of cases require one injection only. In native practice, or when large numbers have to be treated in routine fashion, these drugs may be given intramuscularly, 0.4 grm. dissolved in oil injected into the buttock. The disadvantages are the pain and suppuration which may result.

Even the late tertiary manifestations yield to this treatment. The patient must, of course, be treated on general lines with good food and tonics to improve the health and appetite. Mercury and potassium-iodide mixtures are useful in the tertiary stage. The skin must be kept clean and washed with perchloride-of-mercury solution (1:1,000) daily to allay itching.

P. MANSON-BAHR.

**YELLOW FEVER.**—A specific febrile disease, endemic in parts of tropical America and on the West Coast of Africa, the parasitic cause of which is a leptospira carried and transferred from man to man by the common domestic mosquito, *Stegomyia calopus* (*S. fasciata*) (PLATE 19, Fig. 4, Vol. II, facing p. 250).

**Etiology.**—The germ of yellow fever, according to Noguchi, is the *Leptospira icteroides*, which in its granular form can pass through a Pasteur-Chamberland B bougie, or through a Berkefeld filter. It resembles closely the *Leptospira icterohæmorrhagix*. On the first, second, and third days of the illness the causative germ is circulating in the peripheral blood of the patient; it apparently disappears on the fourth day and, as far as is known, does not reappear during the relapse. Blood taken on those days and injected into non-immunes produces the disease, just as does the filtered blood. If a mosquito of the *Stegomyia calopus* (vel *fasciata*) species bites a yellow-fever patient during the first three days of the illness, the leptospira, which is sucked up with the blood, develops in some way within it during the next twelve or thirteen days. Before this the mosquito is harmless, but now it is capable of transmitting the disease to suitable non-immune individuals. Once infected, the mosquito remains so for at least sixty days (Guiteras), if not for the rest of its life; this observation is important as an indication of the large number of persons an insect may infect. Careful examinations

of the tissues of the mosquito have not thrown any clear light upon the development of the parasite in that situation; but, arguing from the analogy of the malarial cycle in the mosquito, the yellow-fever germ undergoes a development in the tissues of the insect, eventually appearing in some form in the salivary glands, and during the act of biting is transmitted with the saliva to man.

One attack of yellow fever confers immunity for a long period, probably for the rest of life; this was supposed to explain why the indigenous whites and blacks in an endemic area are immune, for they were all believed to have had the disease in a mild and often unrecognized form in their youth. It is certainly the new-comers to a place, the "non-immunes," who acquire the disease and who keep the infection going in the endemic areas. The theory propounded by Boyce, that between epidemics the disease exists in mild unrecognized forms—inflammatory fever—in negroes and others, and that mosquitoes thus obtain their infection, will not hold good for the West India Islands; here, though there is a constant supply of immigrants, the disease may quite disappear for years at a time. This is true of St. Lucia, Barbados, and other islands. To cause its reappearance a fresh introduction is required. A racial immunity has also been suggested, for it has been said that the negro either does not acquire yellow fever or only acquires it in a mild form. How far this is true is uncertain. In some of the Barbados epidemics the negroes have died in considerable numbers. When infection is reintroduced into places where the disease has been stamped out, or at least has been in abeyance for long periods, it is prone to be very severe. This may possibly be explained by the fact that so many young non-immunes have grown up in the meantime. The question of immunity to yellow fever is a very interesting one, and affords a field for further research.

**Pathology.** On examining a case of yellow fever post mortem, one is at once struck with the intense yellow staining of the body; hence the name of the disease. A post-mortem rise of temperature may be noted if the examination is made soon after death. In addition to the yellow coloration, the body often shows post-mortem lividity in a marked degree, and hæmorrhages of varying size, scattered widely over the trunk and limbs. Black fluid is commonly seen oozing from the mouth. On the abdomen being opened, the

## YELLOW FEVER

most striking feature to be noted is the condition of the liver. The organ is of a yellow or yellowish-brown colour, its cut surface being friable and usually exsanguine, though, if death has taken place early, hyperæmia may still be present. This appearance of the liver is very characteristic, and at once distinguishes the condition from malaria and other diseases. Sometimes hæmorrhages may be detected in its substance. The size is about normal. Microscopically, the changes consist of fatty degeneration and necrotic breaking down of the parenchymatous cells, these being most marked in the peripheral zones of the lobules. Some interstitial proliferation has been described by several observers. The gall-bladder generally contains bile, often mixed with blood.

The spleen is acutely congested, soft, but of normal size. The kidneys are not enlarged; they may be congested, and on section the substance shows cloudy swelling and early fatty changes. Hæmorrhages are often seen, either under the capsule or in the cortex or about the pyramids. A definite parenchymatous nephritis may be present.

The stomach is also very characteristic in appearance. The mucosa is much swollen and injected, and larger definite ecchymotic areas are met with scattered over the surface. The viscus is generally full of the black, tarry fluid which, when vomited during life, furnishes the typical black vomit. In fatal cases it is found in the stomach, even though it has not been voided during life. The upper part of the small intestine also contains it in considerable amount.

As regards the chest, the heart shows signs of cloudy swelling, generally with petechial hæmorrhages upon the pericardium or endocardium. The lungs are congested, and may also exhibit small hæmorrhages, either on the pleural surface or scattered throughout the substance.

There is little change observable in the brain, apart from meningeal congestion and the usual small petechial hæmorrhages.

**Symptomatology.**—A striking feature which presents itself to the investigator of an outbreak of yellow fever is the large number of cases met with which do not correspond to the usual book descriptions of the disease. Aberrant or irregular forms are very common in commencing epidemics, and make the initial diagnosis correspondingly difficult. Analysing such cases, one may divide the disease into

four distinct varieties: (1) larval, (2) mild, (3) severe, (4) malignant.

1. The **larval form** is generally missed; indeed, it is difficult to see how it should be diagnosed correctly, unless cases of definite yellow fever are occurring—and even then the diagnosis might easily be a wrong one—a little indisposition, some evanescent fever, perhaps a little vomiting, being all that is present. The importance of cases of this class lies in the fact that mosquitoes may thereby become infected and transmit the disease, which may then assume a severe form.

2. Examples of the **mild form**, again, are not specially characteristic. There is a single paroxysm of fever lasting two to four days, then a rapid fall to normal, and nothing more happens. Slight jaundice, however, may be noticeable, and if the urine is examined carefully a little albumin may be detected after the second day. In other instances slightly more severe, headache, pains in the eyes, and vomiting may occur. These should always arouse suspicion.

3. The **severe form** is that which is usually described in textbooks. The onset of the disease is usually sudden, prodromata as a rule not being pronounced. The malady may start with a rigor or with feelings of chilliness down the back. The temperature rises rapidly to 103° or 104° F., and the patient feels and looks ill. There are pains in the back and limbs and severe frontal headache; the skin of the face becomes flushed, the eyes are injected ("ferrety eye"), and photophobia may be present. The tongue is usually described as being sharp-pointed, with bright-red edges and thickly furred dorsum; appetite is lost, and bilious vomiting may appear early. Epigastric pain has been regarded as of great diagnostic importance. Jaundice appears, according to Sternberg, on the third day, but Guiteras believes it can be detected in the conjunctiva as early as the first morning. Albuminuria commences after forty-eight hours, and the amount of albumin increases till, in the later stages, it may be present in enormous quantities. After remaining high for four days the temperature suddenly falls to 100° F. or lower, the ensuing lull being known as the *remission*. In the milder cases the temperature may not rise again, but in the more severe, after remaining down for some hours even to a day, it ascends again, reaching 104° or 105° F., and the second paroxysm or *relapse* appears. The *pulse*, which in the early stages of the

## YELLOW FEVER

disease was full and bounding, and about 120 in frequency, gradually decreases in rate as the time of the remission comes on, and remains as slow as 40 to 60, when the temperature rises the second time. This phenomenon, known as "Faget's sign," forms, when well marked, one of the surest tests of yellow fever, since it is not associated, as far as is known, with any other ailment. During the second attack, or remission, most of the symptoms of the first paroxysm return. Vomiting becomes severe and is accompanied by great tenderness in the epigastrium; there is thirst, with much prostration; the tongue becomes dry and cracked, and hæmorrhages may appear on different parts of the body. One of two things may now happen: either the temperature eventually falls after two or three days and the patient recovers, or further symptoms appear—black vomit, mæna, suppression of urine, delirium, coma, convulsions—ending in death. In cases which recover, the fall of temperature is often associated with sweating and an increase in the amount of urine. The vomiting ceases, and the albuminuria lessens and finally disappears.

4. In the **malignant form** the symptoms proper to the remission stage make their appearance in the first three or four days of the fever, that is, during the first stage. The temperature may run as high as 105° or 106° F. Violent vomiting followed by black vomit quickly appears, and symptoms referable to the nervous system soon become prominent; such are subsultus tendinum, hiccough, tremors, and delirium. Frequently profuse hæmorrhages (similar to those in black smallpox) appear under the skin, and death quickly takes place.

Sequelæ, such as boils, abscesses, dysentery, and general enfeeblement may follow an attack of yellow fever, but, as a rule, when convalescence begins the progress to recovery is rapid.

**Diagnosis.**—A fever of a single paroxysm accompanied by congestion of the face, ferreted eyes, epigastric pain, gastric irritability, albuminous urine, black vomit, and other hæmorrhages can hardly be mistaken for anything else than yellow fever. Such typical cases, however, as already mentioned, are the exception rather than the rule in many epidemics of this disease.

Without doubt the disease which offers the greatest difficulties in diagnosis is *malignant malaria*, and its bilious remittent form has from time immemorial been mistaken for yellow

fever, and vice versa. In places where malaria exists the slightest suggestion of fever at once results in a large dose of quinine being given, and the very valuable testimony of parasites in the peripheral blood as a means of differentiating between the two complaints is lost. A consideration of the characteristic features of yellow fever that have just been described justifies the opinion that not one of these, taken individually, is sufficient for a reliable and certain diagnosis. Much stress has, for example, been laid on the facial appearance—the flushing of the skin, the injection of the eyes, their ferreted appearance, and the anxious expression. Though this is well marked in certain cases, in others it is not so evident; and in cases of malarial fever, flushing of the face and injection of the conjunctivæ are by no means uncommon.

The condition of the *tongue* presents the same difficulties. Although it is true that in each of these diseases characteristic appearances of this organ may be encountered, namely, the broad, flabby tongue in malaria, and the sharp-pointed tongue with bright-red edges and thickly furred dorsum in yellow fever, yet varying intermediate forms are often met with and render a certain diagnosis impossible.

The *skin* may be hot and dry or moist in both diseases. Guiteras lays stress on the early appearance in yellow fever of jaundice, which, according to him, is noticeable in the conjunctiva on the first day of the disease. Sternberg, however, puts the oncoming of the jaundice as late as the third day. The general yellow colouring of the skin is not constant; in some cases of yellow fever it only appears late or may be absent, whereas in some cases of malaria it is very pronounced. The same may be said of the different forms of purpura which may be found in both diseases.

**Temperature and pulse.**—The relation of the temperature to the pulse is one of importance in distinguishing malaria from yellow fever, the gradual slowing of the latter, even when the temperature is still high, being especially indicative of yellow fever, the rate often sinking as low as 40 beats per minute. This is not seen in malaria.

**Albuminuria.**—Albumin in the urine, which has been considered so diagnostic of yellow fever, is not uncommon in many of the severe types of malaria, and may be present in them in a high degree.

**Epigastric pain.**—This is common in yellow



## YELLOW FEVER

fever, especially on applying slight pressure in the region of the epigastrium, but it may be mistaken for the hepatic pain of a congested malarial liver or for an amœbic hepatitis. *Enlargement of the liver and spleen* cannot alone be relied upon for diagnosis. In some of the acutest cases of malaria they are not enlarged at all, and even though enlargement be found it may only mean that the patient has previously suffered from malaria. Enlargement of the liver and spleen in patients suffering from yellow fever is therefore not uncommon in malarial countries.

*Vomiting.*—Bilious vomiting is an early feature of both diseases. Unfortunately, though rare, vomiting of black material may occur in what are known as the hæmorrhagic forms of malarial fever, and, on the other hand, the black vomit so typical of yellow fever may fail to appear, especially in the mild cases.

*Hæmaturia* and *melæna* occur in both diseases, but hæmorrhages generally are more common in yellow fever than in malaria. Hæmoglobinuria occurs in blackwater fever; never in yellow fever.

It must not be forgotten that in malarial districts the recognition of yellow fever will be attended by difficulties, because many of those attacked will have suffered or still be suffering from malaria. Whenever possible, careful autopsies should be made in all doubtful cases, as this is the only way of completing the diagnosis. The presence of the characteristic malarial pigment, or of parasites localized in the brain, spleen, or gastric mucosa, at once shows that the case is malarial; whereas in yellow fever these are absent, the liver shows the typical yellow colour, and black vomit is found in the stomach.

*Dengue*, another disease which may be mistaken for yellow fever, may be distinguished by the absence of albuminuria and by the preliminary rash and the leucopenia. *Weil's disease* (acute infectious jaundice, spirochæto-sis icterohæmorrhagica) resembles yellow fever closely in some respects, and has sometimes been mistaken for it. The temperature, however, keeps up longer than in yellow fever. A search should be made for the spirochætes in the peripheral blood, but the most certain method is that of inoculating guinea-pigs, when spirochætes will subsequently be found in their blood, liver, and tissues. Other infectious jaundices and *acute yellow atrophy* may in rare instances cause confusion.

**Prognosis.**—The death-rate varies between

30 and 80 per cent. in different epidemics. Yellow fever must always be considered a deadly disease. Excessive albuminuria, great diminution in the amount of urine, high temperature, black vomit, melæna, and other hæmorrhages, hiccough, delirium, are features of grave omen.

**Treatment.**—In the main this must be symptomatic, no specific drug for the disease being known. The patient must be confined strictly to bed in a well-ventilated room under a mosquito curtain and carefully watched. Sternberg devised a mixture which he thought was of value in alleviating the symptoms and in saving life. It consists of sod. bicarb. 150 gr., hydrarg. perchlor.  $\frac{1}{2}$  gr., water to 40 oz. This may be prescribed in doses of 3 tablespoonfuls ( $1\frac{1}{2}$  oz.) every hour. For the vomiting one may apply a mustard-leaf to the epigastrium, or give sips of iced champagne. Chloroform (2 or 3 min.) frequently repeated, tr. iodi (1–2 min.), creosote (1 min.) in mucilage or sugar, from time to time, or nitrate of silver ( $\frac{1}{2}$ – $\frac{3}{4}$  gr.), may be found useful. Opium should not be given at all. Heart failure may be met by stimulants such as alcohol and strychnine. For suppression of urine, dry cupping over the loins, hot fomentations, and hot packs are beneficial; Sternberg's mixture is said to prevent the onset of this symptom, for which Carroll recommends the injection of 15 gr. of urea. Perchloride of iron, acetate of lead, and injection of ergotine have been tried for the black vomiting.

For the first two or three days it is better to give no food. Afterwards, if the temperature is below 102° F., milk and lime water, toast water, albumen water, and barley water may cautiously be tried. Often during the remission the patient may feel comparatively well, but great care must be taken at this time, no increase of diet being allowed. The patient must not exert himself in any way. After the temperature has remained normal for four days the food may be gradually increased, and chicken broth, custard puddings, etc., permitted. Strong beef extracts and strong alcoholic stimulants are injurious. If alcohol is required, iced champagne is the most suitable form in which to administer it.

After convalescence a change of air and scenery is advisable. As one attack confers immunity, there is no danger either from remaining in the epidemic area or from returning to it later.

**Prophylaxis.**—Since we know that yellow

## YELLOW FEVER

fever is spread solely by the *Stegomyia calopus*, it is clear that infection may be prevented by avoiding the bite of this mosquito. If the mosquitoes are numerous, this is not an easy matter, as they are often very active by day, and bite vigorously, especially about midday or earlier. Theoretically, all domestic mosquitoes should be destroyed, and, if this could be done, diseases such as yellow fever, filariasis, and dengue would come to an end. The total destruction of any given genus of mosquito is by no means an easy matter, but that is no reason why campaigns should not be conducted with a view to diminishing their numbers and destroying them as far as possible. The Americans, by an active war against the mosquito, succeeded in stamping out yellow fever in Havana, a place from which, in the days of the Spanish occupation, the disease was never absent. By adopting similar measures the disease was extirpated in the Panama Canal zone, thus permitting of the completion of the canal. The plan of attacking the disease in Havana may be considered here in some detail, as it has provided the classical example on which procedures in other places have been based. When a case of yellow fever was detected the sanitary authorities took immediate steps to render the house in which it had occurred mosquito-proof. No more mosquitoes could get in to bite the patient and so become infected, and any that were already in were carefully destroyed by fumigating with sulphur fumes or pyrethrum, or other methods. Immediately after this was done, the patient could no longer be a source of danger to anyone. At the same time, a general war against the stegomyia and its breeding-places was waged throughout the town, and a great reduction in their numbers quickly effected. In dealing with a suspected case the same procedure should be followed.

Mosquitoes must be prevented from biting the patient, and those which have already had an opportunity of doing so must be destroyed. For personal prophylaxis, every dweller in a yellow-fever area should sleep in a properly fitting mosquito net, and all breeding-grounds of *S. calopus* around the house should be carefully searched out and destroyed. When visiting yellow-fever patients who are not properly protected, it is very important to avoid the bites of mosquitoes. An attendant may prevent them from settling on the back of the neck, a possible place of attack, by waving a towel.

The disinfection of ships is also important, because infected mosquitoes may quite easily be carried by them from one port to another. Ships coming from an infected port should not be allowed to enter another until they have been completely fumigated by the Clayton or other process. The Clayton method consists in filling up the holds and other parts of the ship with sulphur fumes, all mosquitoes thus being killed and any danger from yellow fever avoided. This procedure is essential for every ship that has had yellow fever on board. All quarantine stations should have a series of rooms, carefully mosquito-proofed, to one of which a suspected case can be transferred immediately on arrival. The adoption of these measures has stamped out yellow fever from almost all its old haunts; it now only survives in such localities as parts of Central and South America, where anti-mosquito campaigns have not been instituted.

G. C. Low.

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**ZINC POISONING** (*see* POISONS AND POISONING).

**ZONA** (*see* HERPES ZOSTER).



## INDEX OF DETAILS

- ABDOMINAL reflexes**, iii. 89  
**Abortifacients**, i. 20  
**Abscess of lung**, ii. 225  
     — palmar, iii. 534  
     — prostatic, iii. 9  
**Acanthopis antarcticus**, iii. 221  
**Acarus scabiei**, iii. 149  
**Accessory air sinuses**, diseases of, iii. 198  
     — food factors, iii. 516  
**Accidental hemorrhage**, i. 504  
**Accommodation iridoplegia**, ii. 411  
     — spasm of, iii. 99  
**Accouchement forcé**, ii. 162  
**Accoucheur's hand**, iii. 340  
**Achalasia**, iii. 209  
**Achorion quinckeanum**, ii. 182  
     — schönleinii, ii. 482  
**Acid calcium (stellar) phosphate crystals in urine**, iii. 454  
     — sodium urate in urine, iii. 454  
**Acidity of gastric contents**, i. 518  
**Acladiosis**, iii. 139  
**Acladium castellanii**, iii. 139  
**Acne bacillus**, iii. 172  
     — indurata, i. 33  
     — papulosa, i. 33  
     — punctata, i. 33  
     — pustulosa, i. 33  
**Aconite**, ii. 604  
**Aconitine**, ii. 604  
**Accoumeter**, i. 401  
**Acqui waters**, iii. 225  
**Acriflavine**, iii. 542  
**Acrocephaly**, iii. 210  
**Actinomyces**, staining characters of, i. 146  
**Actinomycosis, mammary**, i. 187  
**Actinotherapy**, ii. 204  
**Addisonian anemia**, i. 57  
**Adipocere**, ii. 618  
**Adolescent kyphosis**, iii. 212  
     — rickets, iii. 138  
     — scoliosis, iii. 210, 211  
**Adrenalin**, administration of, i. 71  
     — composition and physiological action of, iii. 202  
     — therapeutic effects of, ii. 426  
**Afferent system, physiology of**, ii. 371  
**African tie fever**, iii. 99  
**After-pains**, iii. 35  
**Agglutination reactions**, iii. 181  
     — tests after inoculation with typhoid-paratyphoid vaccine, iii. 183  
**Agglutinins in bacillary dysentery**, iii. 183  
**Agglutinins in cholera**, iii. 181  
     — in food poisoning, iii. 183  
     — in plague, iii. 184  
     — in tuberculosis, iii. 184  
     — in undulant fever, iii. 184  
**Agnosia**, i. 97  
**Aix-la-Chapelle waters**, iii. 225  
**Aix-les-Bains waters**, iii. 225  
**Akoria**, iii. 273  
**Albuminuric retinitis**, iii. 110  
**Alcoholic neuritis**, ii. 303  
**Aleppo sore**, ii. 429  
**Algid plague**, ii. 551  
**Alimentary intoxications**, ii. 185  
**Alkalinuria**, iii. 442  
**Allen's diet**, i. 318  
**Allergic reaction in tuberculosis**, iii. 190  
     — in typhoid fever, iii. 192  
**Alloxuric bases in urine**, iii. 450  
**Alpha rays**, iii. 73  
**Alveolar abscess**, i. 23  
**Alypin, administration of**, i. 71  
**Amanita muscaria**, ii. 605  
     — phalloides, ii. 605  
**" Amaurotic cat's eye,"** iii. 115  
**Ambulatory plague**, ii. 551  
**Amino-acids in urine**, iii. 149  
**Ammonia in urine**, iii. 149  
**Ammonium urate in urine**, iii. 154  
**Ammonium magnesium ("triple") phosphate crystals in urine**, iii. 454  
**Anemia pseudo-leukemica infantum**, i. 62  
**Anaesthesia, general, infiltration**, i. 71, 72  
     — local, i. 71  
     — regional, i. 71, 72  
     — spinal, i. 72  
**Anal abscess**, ii. 509  
**Analysis, in psychogenic disorders**, iii. 23  
**Anaphylaxis, and colloids**, i. 270  
**Ancestrodon contortrix**, iii. 221  
     — piscivorus, iii. 221  
**Ancylostoma duodenale**, ii. 126  
**Andiridia**, i. 465  
**Anergic stupor**, iii. 284  
**Angina, Ludwig's**, ii. 222  
**Angular curvature**, iii. 231  
**Anidrosis**, iii. 296  
**Animal irritants**, i. 407  
**Ankle-clonus**, iii. 88  
**Ankle-jerk**, iii. 88  
**Ankle-joint, sprains of**, iii. 251  
**Annular psoriasis**, iii. 17  
     — rickets, ii. 434  
**Anoci-association**, iii. 196  
**Anophelineæ**, ii. 248  
**Anosmia**, iii. 220  
**Anteflexion of uterus**, ii. 497  
**Anterior rhinoscopy**, ii. 391  
**Antero-posterior diameter of pelvic outlet**, ii. 506  
**Anteversion of uterus**, ii. 497  
**Antibacterial serums**, ii. 67, 77  
**Anti-beriberi food factor**, iii. 516  
**Antibodies**, ii. 66  
**Antigenæ**, ii. 65  
**Anti-neuritic food factor**, iii. 516  
**Antipyrin rashes**, i. 380  
**Anti-scorbutic food factor**, iii. 517  
**Antitoxic serums**, ii. 66  
**Antitoxins as colloids**, i. 269  
**Aortic second sound**, iii. 193, 194  
**Apenta waters**, iii. 226  
**Aphthous ulceration**, iii. 277  
**Apoplectic ovum**, ii. 623  
**Appendix dyspepsia**, i. 102  
**Appetite, excessive**, iii. 272  
     — loss of, iii. 272  
**" Apple jelly " papules**, iii. 208  
**Argyria**, ii. 541  
**Arsenic-acid eruptions**, ii. 597  
**Arsenical dermatitis**, i. 380  
     — neuritis, ii. 304  
     — pigmentation, ii. 511  
**Arsenious sulphide**, ii. 597  
**Arseniuretted-hydrogen poisoning**, ii. 597  
**Arseno-argenticum**, iii. 315  
**Arsenobenzol preparations, precautions in administration of**, iii. 318  
     — properties of, iii. 315  
     — toxic effects of, iii. 316  
     — varieties of, iii. 313  
**Arsenobillon**, iii. 313  
**Arsphenamine**, iii. 313  
**Arthritis sèche**, iii. 128  
**Artificial feeding**, ii. 60  
**Ascaris lumbricoides**, ii. 126  
**Aspergillus flavescens**, ii. 189  
     — fumigatus, ii. 489  
     — niger, ii. 189  
**Ateleiosis**, ii. 99  
**Atony, gastric**, iii. 264  
**Atrophic pharyngitis, chronic**, ii. 532  
     — rhinitis, iii. 132  
**Atrophy, muscular, diagnosis of**, ii. 308  
     — optic, ii. 412  
     — peroneal muscular, ii. 309  
**Attic disease**, ii. 441  
**Auchmeromyia luteola**, ii. 318  
**Auditory system, physiology of**, ii. 373

## INDEX OF DETAILS

- Aural, i. 444  
 Auricle, eczema of, ii. 438  
 Auto-intoxication, ii. 484  
 Axillary abscess, i. 24  
 Axis-cylinders, ii. 364
- BACILLARY emulsion, iii. 182**  
*Bacillus* *acnes*, i. 32  
 ——— *anthracis*, i. 91, 140  
 ——— *botulinus*, i. 181  
 ——— *comma*, i. 146, 242  
 ——— *diphtheriae*, i. 146  
 ——— *enteritidis*, ii. 606  
 ——— *fusiformis*, i. 146  
 ——— *influenzae*, i. 146  
 ——— *leprae*, i. 146; ii. 183  
 ——— *mallei*, i. 146, 532  
 ——— *mucosus capsulatus*, iii. 418  
 ——— *oedematis maligni*, i. 146  
 ——— *paratyphosus*, iii. 395, 413  
 ——— *perfringens*, i. 146, 508  
 ——— *pertussis*, iii. 536  
 ——— *pestis*, i. 146; ii. 548  
 ——— *prodigiosus*, i. 100  
 ——— *pyocyaneus*, i. 100  
 ——— *sporogenes*, i. 508  
 ——— *tetani*, iii. 338  
 ——— *tuberculosis*, i. 146  
 "Backdoor splint," iii. 233  
 Bacterial casts in urine, iii. 455  
 Bacteriolytic tests, iii. 185  
 Bacterium *hodgkini*, ii. 237  
 Bacteriuria, i. 308, iii. 61  
 Baden-Baden waters, iii. 224  
 Bagnères-de-Bigorre waters, iii. 223  
 Bagnoles-de-l'Orne waters, iii. 223  
 Balantidium *coli*, i. 383  
 "Bald ringworm," iii. 110  
 Ballottement, internal, iii. 2  
 Bandicoot, ii. 549  
 Bang lamp, ii. 205  
 Banting's diet, ii. 397  
 Barèges waters, iii. 225  
 Barium, ii. 509  
 Bartholinitis, i. 540  
 Bartholin's duct and gland, diseases of, iii. 526, 527  
 Basal ganglia, local lesions of, ii. 361  
 ——— gummatous meningitis, i. 230  
 Bath waters, iii. 223  
 Baths, gaseous thermal, iii. 222  
 ——— simple thermal, iii. 222  
 ——— thermo-mechanical, iii. 222  
 Battaglia waters, iii. 223  
 Beard ringworm, iii. 140, 141  
 Beat-band, ii. 85  
 Beck's bismuth paste injection, iii. 198  
 Bed-bug bites, iii. 262  
 Bell's delirium, i. 278  
 Bence-Jones albumose in urine, iii. 452  
 ——— myelomatosis, ii. 318  
 ——— protein in urine, iii. 452  
 Benedict's test, qualitative, iii. 445  
 Benedict's test, quantitative, i. 345, iii. 447  
 Bengal, ii. 319  
 Benian's modification of Burri's method, iii. 305  
 Benign tertian fever, ii. 248  
 "Bent back of soldiers," ii. 389  
 Berthollet bath, ii. 53  
 Benzidine test for hæmoglobin in urine, iii. 453  
 Beta rays, iii. 73  
 Beta-oxybutyric acid in urine, iii. 445  
 Bex waters, iii. 224  
 Bial's orcin test, iii. 447  
 Bile acids in urine, iii. 454  
 ——— pigments in urine, iii. 453  
 Bilharzia, iii. 158  
 Biliary cirrhosis, ii. 215, 216  
 Bilious typhus fever, iii. 99  
 Bipolar vision, iii. 512  
 Bismuth-feeding, ii. 404  
 Bites and stings of insects, iii. 202  
 Bitis *arietans*, iii. 221  
 "Black dot" ringworm, iii. 140  
 ——— "fever," iii. 141  
 ——— rat, and plague, ii. 540  
 ——— snake, iii. 221  
 ——— tongue, iii. 279  
 Bladder, nervous mechanisms of, iii. 238  
*Blanfordia* *nosophora*, iii. 163  
 Blastomycosis, iii. 207  
 "Bleeders," i. 561  
 Blood mola, iii. 623  
 ——— transfusion, iii. 351  
 Blood-cysts of ovary, ii. 446  
 Blood-urea, ii. 330  
 Body, cooling of, after death, ii. 617  
 Body-louse, ii. 487  
 Bordet-Gengou reaction, iii. 186  
 Boric acid, as an irritant, ii. 600  
 Bottle bacillus, iii. 172  
 Boubas, iii. 564  
 Brachypnura, i. 397  
 Brand's method, ii. 51  
 Brain-stem, local lesions of, ii. 357  
 Breakbone fever, i. 327  
 Breasts, management of, iii. 35  
 Brilliant-green, iii. 542  
 Brissaud's type of infantilism, ii. 100  
 Broad-ligament cysts, ii. 446  
 Brodie's abscess, ii. 436, 437  
 Bromine poisoning, ii. 600  
 Bronchi, foreign bodies in, i. 491  
 Bronchioectasis, i. 194  
 Brown rat, and plague, ii. 519  
 Brunings' laryngoscope and bronchroscope, iii. 107  
 Bubonic plague, ii. 550  
 Budin's method, ii. 92  
 Bullinus *dybowskii*, iii. 159  
 ——— *innesi*, iii. 159  
 ——— *ungarus caruleus*, iii. 221  
 ——— *fasciatus*, iii. 221  
 Bourbonne-les-Bains waters, iii. 224  
 Burri's method, iii. 305  
 Bursitis, i. 204, 205  
 Butter of antimony, ii. 509  
 Buxton waters, iii. 223
- CADAVERIC lividity, ii. 617**  
 Calcification of pericardium, ii. 518  
 Calcium carbonate in urine, iii. 455  
 ——— oxalate in urine, iii. 454  
 ——— sulphate in urine, iii. 455  
 Calliphora, ii. 318, 319  
 ——— erythrocephala, ii. 319  
 ——— vomitoria, ii. 319  
 Calmette's ophthalmic reaction, iii. 101  
*Calymmatobacterium granulomatis*, iii. 418  
 Carabana waters, iii. 226  
 Carbohydrates, chemistry of, ii. 483  
 Carbon compounds, chemistry of, ii. 482  
 Carbonic-acid poisoning, ii. 604  
 Cardiac oedema, ii. 399  
 "Cardiospasm," ii. 405  
 Carditis, iii. 119  
 Care committees, tuberculosis, iii. 393  
 Carlsbad waters, iii. 226  
 Carneous mole, ii. 623  
 Caruncle, examination of, i. 468  
 Caseating tuberculous peritonitis, ii. 528  
 Caseous rhinitis, iii. 132  
 ——— tuberculosis, iii. 40  
 Casts in urine, iii. 455  
 Catarrhal pneumonia, ii. 570  
 Catatonic stupor, iii. 285  
 Catheter life, iii. 7  
 Caustic alkalis, ii. 595  
 Causus *rhombeatus*, iii. 221  
 "Cauterets" waters, iii. 225  
 Cavernous angioma, ii. 331  
 Central pneumonia, ii. 565  
 Cerastes *cornutus*, iii. 221  
 Cercaria, iii. 159  
 Cereæ *flexibilitas*, i. 208  
 Cerebellum, local lesions of, ii. 360  
 Cerebral ataxy, i. 135  
 ——— cortex, focal lesions of, ii. 363  
 Cerebriform nævus, ii. 299  
 Cerebro-spinal rhinorrhœa, iii. 133  
 Certification of insanity, ii. 111  
 Cervical abscess, i. 24  
 ——— glands, enlargement of, ii. 241, 243  
 ——— sympathetic lesions, iii. 509  
 Cervicitis, i. 546  
 "Chapped" lips, ii. 207  
 Chatelguyon waters, iii. 224  
 Cheilitis *exfoliativa*, iii. 173  
 Chemistry, modern, and diagnosis, ii. 486  
 ——— and dietetics, ii. 486  
 ——— and therapeutics, ii. 487

## INDEX OF DETAILS

- Chemosis, i. 281  
 Chimney-sweep's cancer, ii. 87  
 Chloral habit, i. 381  
 Chlorides in urine, iii. 412  
 Chlorine, ii. 605  
 Chloroform, administration of, i. 69  
 ——— mixtures, administration of, i. 70  
 Cholesterol in urine, iii. 455  
 Choluria, iii. 453  
 Chondro-arthritis, i. 117  
 Choroid, sarcoma of, iii. 115  
 Choroidal atrophy, iii. 483  
 Choroiditis, iii. 481  
 ——— anterior, iii. 482  
 ——— central, iii. 483  
 ——— ——— guttate, iii. 483  
 ——— degenerative forms of, iii. 483  
 ——— ——— diffuse, iii. 482  
 ——— ——— disseminated, iii. 482  
 ——— ——— purulent, iii. 482  
 Chromogen in urine, iii. 152  
 Chrysomyia, ii. 319  
 ——— macellaria, ii. 319  
 Chrostek's sign, iii. 311  
 Chyliform ascites, i. 122  
 Chyluria, i. 484  
 Cicatrization, residual, ii. 412  
 Ciliary injection, i. 468  
 Circinate psoriasis, iii. 17  
 Citron's method, i. 315  
 Claviceps purpurea, ii. 605  
 Climate in anæmia, i. 253  
 ——— in asthma, i. 251  
 ——— in bronchitis, i. 254  
 ——— in convalescence, i. 254  
 ——— in dyspepsia, i. 251  
 ——— in gout, i. 254  
 ——— in Graves's disease, ii. 254  
 ——— in hæmoglobinuria, paroxysmal, i. 255  
 ——— in heart disease, i. 255  
 ——— in insomnia, i. 255  
 ——— in laryngitis, i. 255  
 ——— in malaria, i. 255  
 ——— in nephritis, i. 255  
 ——— in neuralgia, i. 155  
 ——— in neurasthenia, i. 255  
 ——— in pneumonia, i. 259  
 ——— in pulmonary tuberculosis, i. 255  
 ——— in rheumatism, i. 259  
 ——— in scrofula, i. 259  
 ——— in skin diseases, i. 259  
 Climatic fever, ii. 246  
 Clitoris, malformations of, iii. 524  
 Coarctation of aorta, ii. 8  
 Coast fever, ii. 246  
 Cobra, iii. 221  
 ——— venom hæmolysis, iii. 180  
 Cocaine habit, i. 381  
 Coko, iii. 565  
 Colic constipation, i. 285  
 Collargol method of demonstrating *Spironema pallidum*, iii. 305  
 Colliquative putrefaction, ii. 618  
 Colloidal gold test, i. 154  
 Colour blindness, i. 273  
 Colubrines, iii. 221  
 Combined ocular palsies, ii. 411  
 Complement, ii. 67, 68  
 ——— deviation of, ii. 69  
 Complement-deviation test, iii. 186  
 ——— ——— in bacterial infections, iii. 188  
 ——— ——— in cerebro-spinal meningitis, iii. 189  
 ——— ——— in glanders, iii. 189  
 ——— ——— in gonococcus infections, iii. 188  
 ——— ——— in helminthiasis, iii. 189  
 ——— ——— in identification of blood-stains, iii. 189  
 ——— ——— in malaria, iii. 189  
 ——— ——— in relapsing fever, iii. 189  
 ——— ——— in syphilis, iii. 189  
 ——— ——— in tuberculosis, iii. 189  
 ——— ——— in typhoid fever, iii. 188  
 ——— ——— in whooping-cough, iii. 189  
 Compression paraplegia, ii. 473  
 Conception, retained products of, iii. 107  
 Concomitant strabismus, iii. 280  
 Confluent smallpox, iii. 116  
 Congestion of lung, ii. 228  
 Congestive dysmenorrhœa, i. 304  
 Conglutination, iii. 184  
 Conjugate, external measurements of, ii. 506  
 ——— ocular palsies, ii. 412  
 ——— true, measurements of, ii. 506  
 Conjunctiva, examination of, i. 468  
 Contracted white kidney, ii. 351  
 Contractions, premature, ii. 17  
 Contrexéville waters, iii. 226  
 Contusions, i. 201, ii. 1  
 ——— from medico-legal standpoint, ii. 111  
 Convergent strabismus, iii. 282  
 Cooper-Hewitt lamp, ii. 205  
 Copper arsenite poisoning, ii. 597  
 ——— poisoning, ii. 599  
 Cordylobia, ii. 319  
 Cornea, burns of, i. 473  
 ——— examination of, i. 468  
 Corneal reflex, iii. 89  
 Corpus luteum, cysts of, ii. 414  
 Corrosive mineral acids, ii. 594  
 Coup de soleil, iii. 290  
 Cowperitis, i. 543  
 Cranio-cerebral injuries, sequelæ of, ii. 4  
 Creatin in urine, iii. 450  
 Creatinin in urine, iii. 450, 455  
 Cremasteric reflex, iii. 89  
 Crotales durissus, iii. 221  
 ——— horridus, iii. 221  
 ——— scutulatus, iii. 221  
 ——— terrificus, iii. 221  
 Croupous pneumonia, ii. 560  
 Crucial ligaments, rupture of, iii. 252  
 Crutch palsy, iii. 247  
 Cryptogenic peritonitis, ii. 522  
 Crystal violet, iii. 542  
 Cycloplegia, ii. 411  
 Cyclothymia, ii. 489  
 Cylindroids in urine, iii. 455  
 Cyst-contents, examination of, i. 55  
 Cystic degeneration, ii. 153  
 ——— of chorion, ii. 624  
 ——— teratomata of ovary, ii. 446  
 Cytoryctes variolæ, iii. 214, 485  
 DABOIA, iii. 221  
 Danyss phenomenon, ii. 66  
 Daphne mezereum, i. 407  
 Dark-ground illumination, technique of, iii. 304  
 Dax waters, iii. 223  
 Dead, identification of, ii. 615  
 Deafness, toxic, ii. 164  
 Death adder, iii. 221  
 Demodex folliculorum, i. 32  
 Dendrites, ii. 364  
 Dental neuralgia, i. 331  
 Dentigerous cysts, i. 331  
 Dermatitis artefacta, i. 408  
 ——— drug, i. 378  
 Dermatobia, ii. 319  
 Dermo lamp, ii. 205  
 Dermoid cysts, examination of contents of, i. 155  
 Detachment of retina, iii. 111  
 Devergie's disease, ii. 547  
 Dextrins in urine, iii. 448  
 Dextrose in urine, iii. 445  
 Diabetes mellitus, chemical pathology of, ii. 486  
 Diabetic retinitis, iii. 111  
 Diacetic acid in urine, iii. 444  
 Diagonal conjugate, measurements of, ii. 506  
 Dial, ii. 602  
 Darsenol, iii. 313  
 Diastase reaction, ii. 310  
 Dibothriocephalus latus, ii. 129  
 Dicephalus, ii. 301  
 Diet, scientific regulation of, ii. 486  
 Diffuse lipoma of neck, ii. 207  
 ——— nephritis, ii. 341  
 ——— psoriasis, iii. 17  
 Dilatation, gastric, acute, iii. 266  
 ——— oesophageal, ii. 405  
 Dinitrobenzene, ii. 602  
 Diphyllbothrium latum, ii. 129  
 Diplococcus rheumaticus, i. 422, 430  
 Dipylidium caninum, ii. 130  
 Disodo-lyargol, iii. 315  
 Dispensary system, and tuberculosis, iii. 388  
 Disseminated myelitis, ii. 317  
 Dissociated personality, ii. 58  
 Diverticula, ii. 405  
 Donne's test for pus in urine, iii. 455

## INDEX OF DETAILS

Dorsal horns, localization of lesions of, iii. 234  
 Dreyer's emulsions, iii. 182  
 Droitwich brine baths, iii. 224  
 Dube, iii. 564  
 Ducrey's bacillus, iii. 421  
 Duct papilloma, i. 190  
 Ductless glands, i. 303, 450, 508, ii. 239, 244, 424, 452, iii. 291  
 Dust-asthma, ii. 586  
 Dust-bronchitis, ii. 586  
 Dust-phthisis, ii. 587  
 Dust-pneumonia, ii. 587  
 Dysostosis, cleido-cranial, i. 253

EARTHY phosphates in urine, iii. 454

Ebstein's diet, ii. 397  
 Echin carinatus, iii. 221  
 Ectopia cordis, ii. 6  
 Edelmann-Galton whistle, i. 102  
 Effluent paths, ii. 374  
 Effluve method, i. 419  
 Ehrlich phenomenon, ii. 66  
 Ehrlich's diazo reaction, iii. 152  
 ——— theory of immunity, ii. 71  
 Einhorn's duodenal tube, ii. 407  
 Elbow-joint, sprains of, iii. 253  
 Elixir hypophysis, ii. 546  
 Elongation of cervix, iii. 460  
 ——— of uvula, iii. 483  
 Emanations, radium, iii. 73  
 Emotive mechanism, ii. 374  
 Enns waters, iii. 225  
 Emulsion colloids, i. 265  
 Encephalocele, ii. 290  
 Enderteritis deformans, i. 107  
 Endodermophyton castellani, iii. 139  
 Endothermy, i. 361  
 Enhydrina valakaden, iii. 221  
 Entamoeba coli, i. 383, 388  
 ——— histolytica, i. 388  
 ——— tetragena, i. 388  
 Enterobius vermicularis, ii. 125  
 Enterocoele, ii. 31  
 Enzymes as colloids, i. 268  
 Epidemic cerebro-spinal meningitis, ii. 287  
 ——— dropsy, ii. 398  
 ——— jaundice of infants, ii. 140  
 ——— parotitis, ii. 305  
 Epidermophyton inguinale, iii. 139  
 Epidermophytosis, iii. 110  
 Epiglottitis, ii. 166, 168, 170, 171, 173, 174, 175, 176, 177, 178  
 Epileptic stupor, iii. 285  
 Epiptoele, ii. 31  
 Epithelial cells in urine, iii. 455  
 Erb's myopathy, ii. 325  
 Ergotism, ii. 605  
 Eristalis tenax, ii. 318  
 Erythrocytes in urine, iii. 455  
 Ether, administration of, i. 61, 66  
 ——— rectal, i. 69  
 ——— habit, i. 381

Ethyl chloride, administration of, i. 66  
 Eupad, iii. 541  
 Eusol, iii. 542  
 Evidence, medical, ii. 274  
 Exanthematous necrosis, ii. 410  
 Extensor jerk, iii. 89  
 External version, iii. 512  
 Extramedullary spinal tumours, iii. 238  
 Extrathecal whitlow, iii. 531  
 Exudative retinitis, iii. 113

FACIO-SCAPULO-HUMERAL atrophy, ii. 326  
 Faecal fistula, iii. 488  
 Faeces, amoebæ in, i. 481  
 ——— bile in, i. 480  
 ——— bilirubin in, i. 480  
 ——— billverdin in, i. 480  
 ——— blood in, i. 481  
 ——— connective tissue in, i. 481  
 ——— fat in, excess of, i. 480  
 ——— fatty-acid crystals in, i. 480, 481  
 ——— hydrobilirubin in, i. 480  
 ——— mucus in, i. 481  
 ——— muscle-fibres in, i. 481  
 ——— ova of intestinal worms in, i. 481  
 ——— pus in, i. 481  
 ——— soap crystals in, i. 480, 481  
 ——— starch granules in, i. 481  
 Faget's sign, iii. 569  
 Familial jaundice, ii. 146  
 ——— optic atrophy, ii. 414  
 Family cholæmia, simple, ii. 117  
 Famine fever, iii. 99  
 Fannia canicularis, ii. 318  
 ——— scalaris, ii. 318  
 Far East, schistosomiasis of, iii. 163  
 Faradic current, reactions to, i. 415  
 Farado-galvanism, i. 419  
 Farey, i. 532  
 Fat in urine, iii. 456  
 Fat-soluble A, iii. 516  
 Fatigue, industrial, ii. 82  
 Fatty-acid crystals in urine, iii. 456  
 Fehling's test for sugar, i. 315, iii. 415  
 Femoral hernia, ii. 33  
 Ferriol's spots, i. 433  
 Fermentation tests, iii. 416  
 Ferments in urine, iii. 454  
 Fibrillation, auricular, i. 137  
 Fibro-casuous tuberculosis, iii. 11  
 Fibroid tuberculosis, iii. 41  
 ——— of peritoneum, ii. 520  
 Fibromata, ovarian, ii. 455  
 Filaria bancrofti, i. 483  
 ——— demarquayi, i. 483  
 ——— loa, i. 483  
 ——— perstans, i. 483  
 Filatow's sign, iii. 152  
 Fimbrial cysts, ii. 447  
 Fingers, infection of, iii. 530  
 ——— osteal infection of, iii. 534

Fingers, tendon-sheaths of, infection of, iii. 532  
 Finsen-Reyn lamp, ii. 205  
 Fish as food, ii. 606  
 ——— poisonous, ii. 605  
 Flea-bites, treatment of, iii. 262  
 Fleishy mole, ii. 623  
 Flexibilitas cerea, i. 208  
 Flexor jerk, iii. 88  
 Flitwick waters, iii. 226  
 Flutter, auricular, i. 141  
 Fly fungus, ii. 605  
 Fœtal heart-beat, iii. 3  
 ——— movements, iii. 3  
 ——— rickets, i. 30  
 Folie brightique, ii. 355  
 Folin and Denis' modification of Shaffer's oxidation method, iii. 415  
 ——— permutit process, iii. 449  
 ——— "turbidity" method, iii. 445  
 Follicular cysts, simple, ii. 444  
 Fontana's method of demonstrating Spiroplasma pallidum, iii. 304  
 Food preservatives, ii. 606  
 Foods, proprietary, ii. 96  
 Formalin estimation of ammonia in urine, iii. 419  
 Forster's operation, ii. 477  
 Freezing, treatment by, i. 206  
 Freud's system, iii. 21  
 Friedrichshall waters, iii. 220  
 Frimley Sanatorium, grades of work at, iii. 55  
 Fructose in urine, iii. 446  
 Fugues, ii. 58  
 Fulminating plague, ii. 551  
 Functional hyperadrenalis-hyperthyroidism, ii. 386  
 Fungi, poisonous, ii. 605  
 Funic souffle, iii. 3  
 Funnel-chest, i. 234

GALACTOCELE, i. 188  
 Galvanic current, reactions to, i. 411  
 Galy, iii. 314  
 Gamma rays, iii. 73  
 Gangosa, iii. 506  
 Gangrenous mediastinitis, ii. 273  
 Gassing, industrial, ii. 85  
 Gastein waters, iii. 223  
 Gastric ulcer, i. 510  
 Gastrogenous diarrhoea, i. 351, 354  
 Gastrophilus, ii. 319  
 Gauvain's "backdoor splint," iii. 233  
 General exfoliative dermatitis, i. 334  
 ——— paralytic stupor, iii. 285  
 Genoud's lamp, ii. 205  
 Gerhardt's test for aceto-acetic acid in urine, iii. 445  
 Gerontoxin, i. 106

## INDEX OF DETAILS

- Gestation, secondary abdominal, ii. 620  
 ———— intraligamentary, ii. 621
- Giant urticaria, iii. 458
- Glioma, i. 218  
 ———— endophytum, iii. 115  
 ———— exophytum, iii. 115
- Globulin test, i. 152
- Glossina morsitans, iii. 367  
 ———— palpalis, iii. 366, 367  
 ———— tachinoides, iii. 367
- Glucose in urine, iii. 445
- Glucuronic acid in urine, iii. 417
- Glycuronic acid in urine, iii. 417
- Gmelin's test, iii. 453
- Gnat-bites, treatment of, iii. 262
- Gonads, ii. 428
- Gonococcus, i. 146
- Goodman and Stern's estimation of albumin in urine, iii. 451
- Gout, chemical pathology of, ii. 485
- Graham's diet, i. 348
- Gram's solution, i. 146
- Granuloma venereum, iii. 418
- Gravid uterus, retroverted, iii. 116
- Greville electric-heat bath, ii. 53
- Grocco's triangle, ii. 551
- Growth food factors, iii. 516
- Guaiac test for hæmoglobin in urine, iii. 153
- Guaiacum test for blood-stains, i. 174
- Gummata, iii. 308
- Gums, hypertrophy of, iii. 278  
 ———— spongy, iii. 278
- Gunshot wounds from medico-legal standpoint, ii. 112
- Günzberg's test for free HCl, i. 517
- Guttate psoriasis, iii. 17
- Gyrate psoriasis, iii. 17
- HÆMATOIDIN in urine, iii. 455
- Hæmin crystals, i. 174
- Hæmoglobin, estimation of, i. 148
- Hæmolytic jaundice, ii. 146
- Hæmorrhage, extradural, ii. 2  
 ———— intracerebral subcortical, ii. 2  
 ———— intradural, ii. 2
- Hæmorrhagic disease of infants, ii. 276  
 ———— peritonitis, ii. 526  
 ———— scarlet fever, iii. 153  
 ———— smallpox, iii. 216
- Haffkine's plague vaccine, ii. 551
- Haldane's method, i. 148
- Hamunâm R'irha waters, iii. 223
- Hand, infections of, iii. 530
- Harrison's sulcus, iii. 136
- Harrogate waters, iii. 224, 226
- Head-louse, ii. 487
- Heat-apoplexy, iii. 290
- Heat-prostration, iii. 290
- Heberden's nodes, ii. 493
- Hegar's sign, iii. 2
- Heller's cold nitric-acid test, iii. 451, 453  
 ———— "ring" test, iii. 451
- Heliophilus pendulus, ii. 318
- Hellouan-les-Bains waters, iii. 225
- Hellvella esculenta, ii. 605
- Hepatic fever, intermittent, i. 591  
 ———— œdema, ii. 300
- Hepatization, grey, ii. 563  
 ———— red, ii. 563
- Hereditary jaundice, ii. 146
- Heroin, poisoning by, ii. 603
- Herpes vulvæ, iii. 526
- Heterochromia, i. 405
- Hey's test for bile acids in urine, iii. 151
- High-frequency current, i. 119
- Hill's intubation tube, ii. 103
- Hippuric acid in urine, iii. 455
- Hirsuties, iii. 203
- Hoffmann's sign, iii. 311
- Homburg waters, iii. 224
- Homogentisic acid, iii. 117
- Hordeolum internum, i. 176
- Hormonic neuroses, ii. 386
- "Hot-cross bun" skull, iii. 210
- Hotechis pseudochis, iii. 221
- "Hour-glass" screens, iii. 71
- Hunger-pain, iii. 273
- Hutchinson's teeth, iii. 311
- Hydatid cysts, examination of contents of, i. 155
- Hydatid of Morgagni, ii. 117
- Hydatidiform mole, ii. 624
- Hydroencephalocoele, ii. 200
- Hydrobilirubin in urine, iii. 152
- Hydrorrhœa, iii. 133
- Hygroma, cystic, i. 308
- Hymenolepis diminuta, ii. 130  
 ———— nana, ii. 130
- Hyperostosis cranii, ii. 182
- Hyperpnea, i. 307
- Hypersecretion, continuous, iii. 269  
 ———— digestive, iii. 268  
 ———— paroxysmal, iii. 269
- Hypertonic saline, iii. 510
- Hypertrophic emphysema, i. 420  
 ———— pharyngitis, ii. 532
- Hypnotism in neurasthenia, ii. 382
- Hypoadrenia, iii. 292
- Hypoderma bovis, ii. 319  
 ———— lineata, ii. 319
- Hypodermic sarcoid, iii. 372
- Hypoglycemia, i. 342
- Hypophysis, ii. 546
- Hystero-epilepsy, ii. 58
- ILEO-COLITIS of infants, i. 350
- Immunization, active, ii. 72  
 ———— passive, ii. 76
- Incompetence, aortic, iii. 491  
 ———— mitral, iii. 496  
 ———— pulmonary, iii. 505  
 ———— tricuspid, iii. 505
- Indigo in urine, iii. 155
- Induction of abortion, i. 21  
 ———— of premature labour, ii. 161
- Infant, new-born, care of eyes of, iii. 36  
 ———— normal gain of weight by, iii. 37
- Infants, premature, food of, ii. 97  
 ———— weaning of, ii. 97
- Infectious Diseases Notification Act, 1889, ii. 394
- Inferior dental nerve, ii. 377
- Infiltration anaesthesia, i. 71, 72
- Inflammatory œdema, ii. 400
- Infundibulum, ii. 516
- Infundibulum, extract of, effects of, ii. 427
- Infundin, ii. 546
- Infusion, saline, ii. 107
- Inguinal hernia, ii. 31
- Insanity of lactation, iii. 32  
 ———— of pregnancy, iii. 32
- Insect bites and stings, treatment of, iii. 262
- Intercrietal diameter of pelvis, ii. 506
- Interference, nervous, physiology of, ii. 369
- Intermittent fever, ii. 246
- Internal version, iii. 513
- Interspinous diameter of pelvis, ii. 506
- Interstitial keratitis, i. 295
- Intertrigo, i. 108
- Intestinal schistosomiasis, iii. 162  
 ———— stasis, i. 285, iii. 515
- Intracutaneous test, iii. 191
- Intramammary abscess, i. 184
- Intramedullary spinal tumours, iii. 239
- Intravenous saline infusion, ii. 407
- Intubation, i. 371
- Involution of uterus, iii. 33, 35
- Iodide eruptions, i. 380
- Iodine test for bile pigments in urine, iii. 151
- Ischemic contracture, Volkmann's, iii. 519
- Ischiopagus, ii. 301
- Iso-glucuronic acid in urine, iii. 116
- JAIL fever, iii. 114
- Jarisch-Herxheimer reaction, iii. 310, 318
- Jaw-jerk, iii. 89
- Jendrassik's method, iii. 88
- Jenner's method, i. 150
- Joint-sprains, iii. 251
- Jungle fever, ii. 216
- Junker's chloroform inhaler, i. 69
- KAPOSI'S disease, iii. 515
- Karell cure, ii. 322
- Kelly's speculum, i. 311
- Keratitis, bullous, i. 296  
 ———— neuro-paralytic, i. 296  
 ———— profunda, i. 295  
 ———— punctata, i. 296; iii. 48, 181



## INDEX OF DETAILS

- Keratitis, sclerosing, i. 205  
 ——— striate, i. 296  
 ——— syphilitic, i. 295  
 Keratomalacia, i. 296  
 Kharsivan, iii. 313  
 Kidney, contracted white, ii. 351  
 Kissingen waters, iii. 224  
 Klumpke's paralysis, iii. 245  
 Knee-jerk, iii. 87  
 Knee-joint, sprains of, iii. 252  
 Knock-knee, i. 529  
 Koilonychia, ii. 333  
 Koplik's spots, ii. 268, 271  
 Korsakoff's syndrome, i. 42  
 Kraiss, iii. 221  
 Krause's method of labour induction, ii. 162  
 Kreuznach waters, iii. 224  
 Kromayer lamp, ii. 205
- LA BOURBOULE waters, iii. 226  
 Labour, fibroids and, iii. 465  
 ——— ovarian tumours and, ii. 448  
 Labyrinthine nystagmus, ii. 395  
 Lachosis lanceolatus, iii. 221  
 Lachrymal abscess, i. 314  
 ——— gland, i. 467  
 ——— passages, i. 468  
 Lactalbumin, ii. 96  
 "Ladder" diet, i. 348  
 Lævulose in urine, iii. 446  
 Lake's tubinotome, ii. 337  
 Laminectomy, iii. 244  
 Landouzy-Dejerine atrophy, ii. 326  
 Lange's colloidal gold reaction, i. 270  
 Lardacein, i. 53  
 Large-celled splenomegaly, i. 62  
 Larval pneumonia, ii. 570  
 Late rickets, iii. 138  
 Latent squint, iii. 283  
 Lathyrus cicera, ii. 605  
 ——— sativus, ii. 605  
 Leather-bottle stomach, iii. 272  
 Leishmania tropica, ii. 429  
 Leishman's method, i. 150  
 Leptospira icteroides, iii. 509  
 Leucin in urine, iii. 455  
 Leucocytes, enumeration of, i. 150  
 ——— in urine, iii. 455  
 Leukæmic retinitis, iii. 111  
 Leveco waters, iii. 226  
 Lichen ruber acuminatus, ii. 517  
 Lienteric diarrhoea, i. 352, 351  
 Lightning pains, iii. 328  
 Lillingston's apparatus for artificial pneumothorax, iii. 57  
 Lipoids, chemistry of, ii. 483  
 Lipoma of neck, diffuse, ii. 207  
 Lithopædion, ii. 620  
 Littre's hernia, ii. 31  
 Liver poisons, ii. 601  
 Manderindod Wells waters, iii. 225  
 Localization of cerebral lesions, ii. 357  
 ——— of spinal-cord lesions, iii. 234
- Lochia, changes in, iii. 33  
 Loculation syndrome, iii. 239  
 Longitudinal reaction, i. 415  
 Lorain's type of infantilism, ii. 100, 545  
 Lortet's lamp, ii. 205  
 Luchon waters, iii. 225  
 Lucilia, ii. 319  
 ——— casar, ii. 319  
 Ludwig-Malkowski estimation of uric acid in urine, iii. 450  
 Luetin reaction, iii. 191  
 Lumbo-sacral plexus, injury of, iii. 247  
 Luminal, ii. 602  
 Lupus non-exodens, iii. 209  
 Lymphæmia, ii. 192  
 Lymphangitis, i. 484  
 Lymphogranulomatosis maligna, ii. 237  
 Lymphoid leukæmia, acute, ii. 192  
 ——— chronic, ii. 194  
 Lymphomatosis, ii. 237
- MACACUS rhesus, iii. 142  
 Maclean's researches on nephritis, ii. 339, 341  
 ——— urea concentration test, ii. 340  
 Macrocheilia, ii. 207  
 Macrostoma, ii. 207  
 Maddox rods, iii. 283  
 Magnesium phosphate in urine, iii. 455  
 Malassezia tropica, iii. 345  
 Malignant purpuric fever, ii. 287  
 ——— tertian fever, ii. 249  
 Malum coxæ senilis, ii. 433  
 Mammary abscess, i. 184  
 ——— actinomycosis, i. 187  
 ——— cysts, i. 188  
 Mania a potu, i. 41  
 Manson's stain, i. 147  
 Marienbad waters, iii. 226  
 Marriage of epileptics, i. 448  
 Marsh fever, ii. 246  
 Marshall & Woods' lamp, ii. 205  
 Massive exudation in retina, iii. 113  
 ——— pneumonia, ii. 565  
 Mastication palsy, iii. 362  
 Matlock waters, iii. 223  
 Meatus, external, eczema of, ii. 438  
 ——— furunculosis of, ii. 439  
 Median-nerve paralysis, iii. 247  
 Medico-legal cases, post-mortem examinations in, ii. 614  
 ——— reports on, ii. 274  
 ——— standpoint, injuries from, ii. 109  
 Medinal, ii. 602  
 Mégaspores, iii. 139  
 Melanotic carcinoma, iii. 204  
 ——— sarcoma, iii. 204  
 Membranous catarrh of intestines, i. 260
- Membranous dysmenorrhœa, i. 394  
 ——— rhinitis, iii. 181  
 Meningism, ii. 104  
 Meningococcus, i. 146  
 Meningo-encephalitis, vertical, i. 230  
 Meningo-myelocèle, iii. 230  
 Menstrual fistula, iii. 488  
 Mercurial preparations in syphilis, iii. 319  
 ——— methods of administering, iii. 319  
 ——— toxic effects of, iii. 320  
 Mercurialism, i. 380  
 Metabolism, disorders of, ii. 485  
 Metamorphopsia, iii. 482  
 Metchnikoff's theory of immunity, ii. 70  
 Methylene-blue, i. 147  
 Micrococcus catarrhalis, i. 146  
 ——— melitensis, i. 146; iii. 423  
 ——— paramelitensis, iii. 423  
 ——— paratetrigenus, i. 146  
 ——— tetrigenus, i. 146  
 Microdes, iii. 139  
 Micropsia, iii. 482  
 Microsporon furfur, iii. 315  
 Microstoma, ii. 207  
 Middle-ear disease, acute, ii. 439  
 ——— chronic, ii. 441, 442  
 Miliary aneurysm, i. 223  
 ——— tuberculosis of skin, iii. 205  
 Milk, as food, ii. 608  
 ——— condensed, ii. 96  
 ——— cow's, ii. 91  
 ——— dried, ii. 96  
 ——— human, ii. 90  
 ——— modifications of, ii. 93  
 ——— pasteurization of, ii. 92  
 ——— peptonized, ii. 96  
 ——— poisoning by, ii. 608  
 ——— sterilization of, ii. 91  
 Miller lamp, ii. 205  
 Mineral waters, alkaline, iii. 225  
 ——— arsenical, iii. 226  
 ——— calcareous, iii. 226  
 ——— chalybeate, iii. 226  
 ——— indifferent, iii. 223  
 ——— mixed chloride, iii. 223  
 ——— simple, iii. 223  
 ——— sulphated, iii. 226  
 ——— sulphur, iii. 224  
 Miner's beat-elbow, ii. 85  
 ——— beat-knee, ii. 85  
 Minimum hæmolytic dose, iii. 187  
 Modified smallpox, iii. 217  
 Mongolian idiocy, i. 305  
 Monilia albicans, iii. 254  
 Mont Dore waters, iii. 227  
 Moon's teeth, iii. 312  
 Moor baths, ii. 51  
 Morant Baker cysts, ii. 432  
 Morax-Axenfeld bacillus, i. 283  
 Morbilli sine morbillis, ii. 260  
 Moro's test, iii. 191  
 Morphinomania, i. 381

## INDEX OF DETAILS

- Mosquito bites, treatment of, iii. 262  
 Mosquitoes of dengue, i. 328  
   — of filariasis, i. 483  
   — of malaria, ii. 248  
   — of yellow fever, iii. 567, 571  
 Motor ataxy, i. 135  
 Mucocoele, iii. 199  
 Mucosa-membranous entero-colitis, i. 260  
 Mucor racemosus, ii. 489  
 Mucus in urine, iii. 455  
 Müllerian cysts of vagina, iii. 487  
 Müller's exercises, ii. 307  
 Multilocular adenomatous cysts, ii. 444  
 Multiple serositis, ii. 612  
 Mummification, ii. 619  
 Murmurs, diastolic, iii. 493, 494  
   — mitral, iii. 493  
   — presystolic, iii. 494  
 Musca domestica, ii. 318  
 Muscle-sprain, iii. 251  
 Mushrooms, poisoning by, ii. 594  
 Mussels, poisoning by, ii. 605  
 Mydalin, ii. 606  
 Myelæmia, ii. 190  
 Myeloid leukæmia, ii. 193  
 Myelocytosis, ii. 187  
 Myelogenous leukæmia, ii. 190  
 Myelomatosis, Bence-Jones, ii. 318  
 Myoneural junction, ii. 366  
 Myotonic reaction, i. 116  
  
 N.EVO-LIPOMA, ii. 331  
 Nervus cerebriform, ii. 299  
   — lipomatodes, ii. 299  
   — papillomatosis, ii. 299  
   — pilosus, ii. 299  
   — verrucosus, ii. 299  
 Nail, removal of, incisions for, iii. 532  
 Nails, ringworm of, iii. 140  
 Naja bungarus, iii. 221  
   — flava, iii. 221  
   — haja, iii. 221  
   — tripudians, iii. 221  
 Nantwich brine baths, iii. 224  
 Narcotic alkaloids, administration of, i. 71  
 Natal sore, iii. 510  
 Nauheim system, ii. 51  
   — treatment, iii. 101, 224  
   — waters, iii. 224  
 Necator americanus, ii. 128  
 Neisser-Wechsberg phenomenon, ii. 68  
 Neo-arsphenamine, iii. 314  
 Neodiarsenol, iii. 314  
 Neo-endothrix, iii. 139  
 Neokharsivan, iii. 314  
 Neosalvarsan, iii. 314  
 Nerve impulse, ii. 365  
   — leprosy, ii. 185  
 Nerve-cell body, ii. 365  
 Nerve-fibres, centrifugal, ii. 365  
 Nerves, centripetal, ii. 365  
 Nesokia bengalensis, ii. 549  
  
 Neurin, ii. 606  
 Neuro-fibromatosis, general, ii. 384  
 Neurone, ii. 364  
   — physiological, ii. 365  
 Neuro-paralytic keratitis, i. 296  
 Neuro-recurrences, syphilitic, iii. 318  
 Night-sweats in pulmonary tuberculosis, iii. 44, 58  
 Nipples, care of, ii. 88  
 Nitrogen output in urine, total, iii. 448  
 Nitrous oxide, administration of, i. 65  
 Nodular circumscribed lipomatosis, ii. 207  
   — leprosy, ii. 184  
 Nodules, subcutaneous, iii. 120  
 Noise deafness, ii. 164  
 Noises in ear, iii. 345  
 Norwegian rat, and plague, ii. 549  
 Notechis scutatus, iii. 221  
 Novarsenobenzol, iii. 314  
 Novarsenobillon, iii. 314  
 Novocain, administration of, i. 71  
 Nuclein bases in urine, iii. 150  
 Nummular psoriasis, iii. 17  
 "Nutmeg liver," iii. 501  
 Nux vomica, poisoning by, ii. 604  
  
 OBTURATOR hernia, ii. 34  
 Oclusio pupillæ, iii. 479  
 Occupational mortality, ii. 86  
 Ocular nystagmus, ii. 395  
 Oertel's diet, ii. 397  
 Esophageal spasm, ii. 405  
 Esophagus, rupture of, ii. 405  
 Estrus, ii. 319  
 Oidium albicans, iii. 277  
 Oil of bitter almonds, poisoning by, ii. 601  
 Oligæmia, iii. 195  
 Onychogryphosis, ii. 333  
 Onychorrhexis, ii. 333  
 Onychoschizia, ii. 333  
 Opacities, corneal, i. 295  
 Ophthalmic reaction, Calmette's, iii. 191  
 Ophthalmoplegic migraine, ii. 410  
 Oppenheim's sign, iii. 89  
 Opsonic tests, iii. 180  
 Opsonins, ii. 68  
 Orbit, examination of, i. 466  
 Osler's spots, i. 433  
 Ovarian cysts, examination of contents of, i. 155  
   — — — rupture of, ii. 451  
   — — — dermoids, ii. 446  
   — — — extract, effects of, ii. 428  
   — — — insufficiency, i. 51  
 Ovary, prolapse of, ii. 497  
 Oxalic acid in urine, iii. 414  
 Oxyuria vermicularis, ii. 125  
  
 PACKS, ii. 53  
 Palatal reflex, iii. 89  
 Palmar abscesses, iii. 534  
  
 Palpebral aperture, i. 467  
 Paludism, ii. 246  
 Pancreatic cysts, examination of contents of, i. 155  
   — reaction in urine, iii. 448  
 Papulo-necrotic tuberculides, iii. 372  
 Paralysis, diphtheritic, i. 366, 372  
   — of accommodation, iii. 99  
 Paralytic kyphosis, iii. 212  
 Parangi, iii. 504  
 Parathyroid medication, ii. 425  
 Paronychial whitlow, iii. 530, 532  
 Paroxysmal neuroses, ii. 385  
 Parrot's nodes, iii. 210  
 Partial reaction of degeneration, i. 416  
 Pasteurization of milk, ii. 92  
 Pediculus capitis, ii. 487  
   — corporis, ii. 487  
   — pubis, ii. 488  
   — vestimenti, ii. 487  
 Pellagrosin, ii. 489  
 Pemphigus, congenital, ii. 509  
 Penicillium glaucum, ii. 189  
 Peptic ulcer, i. 510  
 Perforating ulcer, iii. 328, 410  
 Perforation in typhoid fever, iii. 403  
 Pericardial effusion, clinical signs of, ii. 509  
 Pericellular cirrhosis, ii. 210  
 Perinephric abscess, ii. 519  
 Perineum, rupture of, ii. 159  
 Periosteal aplasia, ii. 134  
 Peritubal hamatocoele, ii. 621  
 Persuasion, iii. 23  
 Pfeiffer's reaction, ii. 67, iii. 185  
 Pharynx, foreign bodies in, i. 491  
 Phenol, poisoning by, ii. 595  
 Phenyl-hydrazine, poisoning by, ii. 602  
 Phlebotomus, ii. 429  
 Phlyctenular ulcers, i. 292  
 Phloersa, iii. 221  
 Phosphates in urine, iii. 442  
 Phosphorus necrosis, ii. 419  
 Photopsia, iii. 482  
 Phthisis bulbi, iii. 480  
 Physopsis africana, iii. 159  
 Picric acid, ii. 602  
 Pingicula, i. 285  
 Pituglandol, ii. 546  
 Pituitary-gland preparations, effects of, ii. 427  
 Pituitrin, ii. 546  
 Planorbis boissyi, iii. 162  
   — centrinetralis, iii. 162  
   — olivaceus, iii. 162  
 Plantar reflex, iii. 89  
 Plasmodium falciparum, ii. 248  
   — malaria, ii. 248  
   — vivax, ii. 248  
 Plexiform neuroma, ii. 384  
 Plombières douche, ii. 52  
   — waters, iii. 223  
 Pluriglandular therapy, ii. 428  
 Pneumococcal peritonitis, ii. 527  
 Pneumococcus, i. 146, ii. 502

# INDEX OF DETAILS

Poisoning, suspected, conduct of medical practitioner in, ii. 504  
 — — — post-mortem examinations in, ii. 615  
 Poisons, sale of, regulations for, ii. 592  
 Politzer's method, i. 400  
 Polyarticular gout, iii. 120  
 Polycythæmia vera, i. 307  
 Popliteal abscess, i. 27  
 Portal cirrhosis, ii. 210  
 Port-wine mark, ii. 331  
 Postanæsthetic pneumonia, ii. 570  
 Postclimacteric bleeding, ii. 203  
 Posterior interspinous diameter of pelvis, ii. 506  
 Postnasal space, examination of, ii. 391  
 Postoperative pneumonia, ii. 570  
 Postural mechanism, ii. 374  
 Potain's aspirator, ii. 558  
 Potassium bichromate, poisoning by, ii. 509  
 Precipitins, ii. 69  
 — — — bacterial, iii. 185  
 — — — serum, ii. 69, iii. 185  
 Preparation for operations, ii. 408  
 Prepatellar abscess, i. 28  
 Preretinal hæmorrhage, iii. 112  
 Primary sore, characters of, iii. 301  
 — — — — — course of, iii. 302  
 — — — — — diagnosis of, iii. 303  
 — — — — — treatment of, iii. 322  
 Primula obconica, i. 407  
 Progressive myocardial degeneration, ii. 320  
 Prolapse of ovary, ii. 497  
 — — — of uterus, ii. 495, 499, 501  
 Proliferate peritonitis, ii. 525  
 Pronator jerk, iii. 89  
 Propenal, ii. 602  
 Proptosis, i. 406  
 Prostatic abscess, iii. 9  
 Prostatitis, i. 544  
 Proteins, chemistry of, ii. 182  
 Pseudo-broad-ligament cysts, ii. 447  
 Pseudo-chylous ascites, i. 122  
 Pseudo-flatulence, i. 486  
 Pseudo-glioma, iii. 115  
 Pseudo-hypertrophic myopathy, ii. 325  
 Pseudo-lavulose in urine, iii. 116  
 Pseudo-mucinous cysts of ovary, ii. 411  
 Psilosis pigmentosa, ii. 188  
 Puerperal sapremia, iii. 28  
 — — — scarlet fever, iii. 153  
 — — — septicæmia, iii. 29  
 Pulmonary stenosis, ii. 7  
 Punctate psoriasis, iii. 17  
 Pupils, inequality of, i. 469  
 — — — reaction of, to light, i. 469  
 Purdy's test for albumin in urine, iii. 451  
 Pus in urine, iii. 453  
 Pyæmia, puerperal, iii. 27

QUARTAN fever, ii. 249  
 Quickening, iii. 2  
 RADICULITIS, i. 229  
 Rag-picker's disease, i. 91  
 Rammstedt's operation, iii. 67  
 Rats, and plague, ii. 549  
 Rectum, foreign bodies in, i. 492  
 — — — nervous mechanism of, iii. 238  
 Recurrent chancre, iii. 301  
 Red corpuscles, enumeration of, i. 149  
 Red-light treatment of smallpox, iii. 220  
 Re-education of paralysed limbs, iii. 244  
 — — — psychotherapeutic, iii. 24  
 Regional anæsthesia, i. 71, 72  
 Reichenhall waters, iii. 224  
 Remittent icteric fever, iii. 99  
 Renal oedema, ii. 309  
 — — — pericarditis, ii. 517  
 Repair, essentials of, iii. 539  
 Residual cicatrization, ii. 442  
 Retinitis, iii. 110  
 — — — albuminuric, iii. 110  
 — — — circinata, iii. 113  
 — — — diabetic, iii. 111  
 — — — exudative, iii. 113  
 — — — from exposure to bright light, iii. 111  
 — — — leukæmic, iii. 111  
 — — — pigmentosa, iii. 114  
 — — — proliferans, iii. 113  
 — — — purulent, iii. 111  
 Retromammary abscess, i. 185  
 Retro-uterine hæmatocele, ii. 621  
 "Return cases" of scarlet fever, iii. 156  
 Revaccination, iii. 484, 486  
 Reverdin's skin-grafts, iii. 512  
 "Rheumatic fever," iii. 117  
 — — — myocarditis, ii. 323  
 — — — pericarditis, ii. 511  
 Rhinitis, hypertrophic, ii. 335  
 — — — sicca, iii. 132  
 Rhus dermatitis, i. 407  
 Richter's hernia, ii. 31  
 Rickettsia prowazeki, iii. 414  
 Rickety kyphosis, iii. 212  
 — — — scoliosis, iii. 240, 242  
 Rinné's test, i. 402  
 Rock fever, iii. 423  
 Roncegno waters, iii. 226  
 Rosenbach's modification of Gimelin's test, iii. 454  
 Rotch's sign, ii. 509  
 Rothera's modification of Legal's test, iii. 414  
 Royat-les-Bains waters, iii. 225  
 Rubecola sine catarrho, ii. 269  
 — — — sine exanthemate, ii. 269  
 Rubinat waters, iii. 226  
 Ruhemann's ureometer, iii. 450  
 Rupoid psoriasis, iii. 17  
 Russian bath, ii. 53  
 Russo's methylene-blue test, iii. 407

SACHS-GEORGI syphilis reaction, iii. 188  
 Sahli's hæmoglobinometer, i. 149  
 St. Vitus's dance, i. 248  
 Sale of poisons, regulations for, ii. 592  
 Salicyl-sulphonic acid test, iii. 451  
 Nalles d'aspiration, iii. 227  
 Salpingitis, i. 546  
 Salisbury's diet, ii. 397  
 Salso Maggiore waters, iii. 224  
 Salts, inorganic, chemistry of, ii. 483  
 Salvarsan, provocative injection of, iii. 188  
 Salvarsan-natrium, iii. 314  
 Sand, intestinal, ii. 125  
 Sarcophaga carnaria, ii. 319  
 Sarcoptes scabiei, iii. 149  
 Satiety, absence of, iii. 273  
 — — — rapid, iii. 273  
 Sausages, poisoning by, ii. 607  
 Scapulo-humeral type of atrophy, ii. 325  
 Scarlatina, iii. 150  
 — — — anginosa, iii. 153  
 — — — sine eruptione, iii. 153  
 Scarlet-red, iii. 542  
 Schäfer's method of artificial respiration, iii. 106  
 Scheele's green, ii. 597  
 Schinznach waters, iii. 225  
 Schiötz tonometer, i. 534  
 Schistosoma hematobium, iii. 158, 159  
 — — — — — ova of, iii. 159  
 — — — japonicum, iii. 163  
 — — — — — ova of, iii. 163  
 — — — mansoni, iii. 162  
 — — — — — ova of, iii. 162  
 Schlangenbad waters, iii. 223  
 Schwalbach waters, iii. 226  
 Screens for radium apparatus, iii. 73  
 Scrotal cancer, ii. 87  
 Scurf, iii. 172  
 Scurvy, infantile, iii. 169  
 Seborrhæic alopecia, i. 45  
 Seclusio pupillæ, iii. 479  
 Sedimentation method, iii. 182  
 Sensory ataxy, i. 135  
 — — — paths in spinal cord, iii. 235  
 Septicæmia, puerperal, iii. 29  
 Septicæmic plague, ii. 551  
 Septum, nasal, deflections of, ii. 337  
 — — — — — spurs and crests of, ii. 338  
 Serum, anticholeraic, ii. 78  
 — — — antidysenteric, ii. 77  
 — — — antigenococcic, ii. 77  
 — — — antimeningococcic, ii. 77  
 — — — antiplague, ii. 78  
 — — — antipneumococcic, ii. 78  
 — — — — — polyvalent, ii. 77  
 — — — antistaphylococcic, ii. 77  
 — — — antistreptococcic, ii. 77  
 — — — antituberculous, ii. 78  
 — — — antityphoid, ii. 78

## INDEX OF DETAILS

- Serum diagnosis of paroxysmal hæmoglobinuria, iii. 186  
 — dilutions, iii. 182  
 — in poliomyelitis, ii. 78  
 — in scarlet fever, ii. 78  
 — normal, ii. 67  
 Serum-precipitins, ii. 69  
 Shadow test, iii. 92  
 Shellfish, poisoning by, ii. 607  
 Shiga bacillus, i. 381  
 Sialodochitis fibrosa, iii. 121  
 Sick-room, disinfection of, i. 372  
 Silbersalvarsan, iii. 315  
 Silicosis, ii. 588  
 Silver nitrate, ii. 599  
 Sinusoidal current, i. 419  
 Skatol in urine, iii. 444  
 Skin, miliary tuberculosis of, iii. 205  
 Skinner's mask, i. 70  
 " Sleeve " screens, iii. 71  
 Snellen's test-types, i. 471, iii. 94  
 Sodium arsenite, poisoning by, ii. 507  
 — salvarsan, iii. 311  
 Spa waters, iii. 220  
 Spasmodic dysmenorrhœa, i. 302  
 — hour-glass stomach, iii. 266  
 Spasticity, treatment of, iii. 244  
 Spectra of blood, i. 175  
 Spermatozoa, dimensions of, iii. 176  
 Spes phthisica, iii. 46  
 Spider naevi, ii. 331  
 Spina bifida occulta, iii. 230  
 Spinal anæsthesia, i. 72  
 — meningocœle, iii. 230  
 Spirillar fever, iii. 99  
 Spirochæta carteri, iii. 100  
 — duttoni, iii. 100  
 — icterohæmorrhagiae, ii. 144  
 — novyi, iii. 100  
 — obermeieri, iii. 100  
 — pallida (*see* *Spiro-nema pallidum*)  
 — recurrentis, iii. 100  
 Spirochætal fever, iii. 99  
 Spirochætosis icterohæmorrhagica, ii. 144  
 Spiro-nema pallidum, characters of, iii. 299  
 — — — identification of, iii. 304  
 — — — pertenuæ, iii. 565  
 Splenomedullary leukaemia, ii. 190  
 Spore of Malassez, iii. 172  
 Sporotrichosis, iii. 207  
 Sprain-fracture, iii. 251  
 Stains, blood, i. 173  
 — seminal, iii. 176  
 Staphylococcus, i. 146  
 Stegomyia calopus, iii. 567, 571  
 — fasciatus, iii. 567, 571  
 Sterilization of milk, ii. 91  
 Sternberg's mixture, iii. 570  
 Stiffnecked fever, i. 327  
 Stomatitis, gangrenous, iii. 278  
 — gonorrhœal, iii. 277  
 Stomatitis, mercurial, iii. 278  
 — streptococcal, iii. 277  
 Strathpeffer waters, iii. 225  
 Streptobacillus pellagræ, ii. 489  
 Streptococcus, i. 146  
 — erysipelatis, i. 453  
 Subacute combined degeneration, i. 275  
 Subcutaneous nodules, iii. 120  
 — test in tuberculosis, iii. 191  
 — — whitlow, iii. 530, 532  
 Subcuticular whitlow, iii. 530, 532  
 Subhyaloid hæmorrhage, iii. 112  
 Submaxillary abscess, i. 241  
 Subtertian fever, ii. 249  
 Subungual whitlow, iii. 530  
 Sugar, estimation of, in urine, iii. 417  
 Sulfarsenol, iii. 311  
 Sulfoxylate, iii. 315  
 Sulphates in urine, iii. 413  
 Sulphonal, poisoning by, ii. 602  
 Sun-traumatism, iii. 290  
 Superior maxillary nerve, ii. 377  
 Suppurative mediastinitis, ii. 272  
 — pericarditis, ii. 514  
 Supramammary abscess, i. 185  
 Surgical emphysema, i. 421  
 — scarlet fever, iii. 153  
 Suspension colloids, i. 265  
 Suture of tendons, methods of, iii. 331  
 Swabs, i. 147  
 Sydenham's chorea, i. 248  
 Sylvester's method, iii. 105  
 Sympathetic neuroses, ii. 385  
 — — — circulatory, ii. 385  
 — — — gastro-intestinal, ii. 385  
 Synapse, ii. 366  
 Syncephalus, ii. 301  
 Synecioma, i. 250  
 Synecchia, anterior, i. 293  
 — posterior, iii. 478, 480  
 — ring, iii. 479  
 — total posterior, iii. 479  
 Syphilides, iii. 305  
 Syringo-mycelocœle, iii. 230  
 TAbes, gastric crises of, iii. 276  
 Tachypnoea, i. 396  
 Tænia echinococcus, ii. 38  
 — — — mediocancellata, ii. 128  
 — — — saginata, ii. 128  
 — — — solium, ii. 129  
 Talipes calcaneus, iii. 333  
 — equino-varus, iii. 331  
 — equinus, iii. 332  
 — paralytic, iii. 333  
 Tapeworm infections, ii. 128, 606  
 Tarry stools, ii. 276  
 Tenderness, distinguished from pain, i. 5  
 Tendon-jerks, iii. 87  
 Teratoma of ovary, solid, ii. 157  
 Terminal pneumonia, ii. 569  
 Testicular extract, effects of, ii. 428  
 Test-meals, i. 516  
 — fractional, i. 519  
 Tetronal, ii. 602  
 Thecal whitlow, iii. 530, 532  
 Thenar eminence, abscess of, iii. 534  
 Thermic fever, iii. 290  
 Thiersch's superficial skin grafts, iii. 540  
 Thoma hæmocytometer, i. 119  
 Thoracopagus, ii. 301  
 Thoracoplasty, iii. 57  
 Thrombosis, lateral-sinus, ii. 180  
 — of cavernous sinus, septic, ii. 423  
 — of central vein of retina, iii. 112  
 Thymus asthma, ii. 245  
 — — — extract, ii. 428  
 Thyroid medication, ii. 424  
 Tinea alba, iii. 130  
 — — — albigena, iii. 130  
 — — — barbae, iii. 110  
 — — — circinata, iii. 111  
 — — — cruris, iii. 139  
 — — — imbricata, iii. 139  
 — — — intersecta, iii. 139  
 — — — marginata, iii. 139, 141  
 — — — nigro-circinata, iii. 139  
 — — — tonsyosia, iii. 140  
 — — — tonsurans, iii. 138  
 — — — tropicalis, iii. 139  
 Tinned fish, ii. 608  
 — — — foods, ii. 605, 607  
 — — — meat, ii. 606  
 Toluylene-diamine, ii. 602  
 Tonus, physiology of, ii. 369  
 Töpfer's test, i. 517  
 Torsion of pedicle of ovarian tumours, ii. 449  
 Toxæmia, alimentary, i. 43  
 Toxic idiopathy, ii. 72, 101  
 — — — œdema, ii. 399  
 — — — pneumonia, ii. 570  
 — — — vomiting, iii. 267  
 Trachea, foreign bodies in, i. 401  
 Tracheotomy, i. 371  
 Transillumination, iii. 201  
 Transverse diameter of pelvic outlet, ii. 506  
 — — — myelitis, acute, ii. 316  
 Trench gums, iii. 68  
 Treponema pallidum (*see* *Spiro-nema pallidum*)  
 Tribondeau's modification of Fontana's method, iii. 304  
 Trichina spiralis, iii. 359  
 Trichinella spiralis, iii. 359  
 Trichocephalus dispar, ii. 126  
 Trichomonas vaginæ, iii. 176  
 Trichophyton acuminatum, iii. 139, 140  
 — — — albicans, iii. 139  
 — — — blanchardi, iii. 139  
 — — — ceylonense, iii. 139  
 — — — crateriforme, iii. 139  
 — — — ecto-endothrix, iii. 138  
 — — — endo-ectothrix, iii. 139  
 — — — endothrix, iii. 138, 139  
 — — — equinum, iii. 139

## INDEX OF DETAILS

*Trichophyton felineum*, iii. 139  
 — *gypseum*, iii. 139  
 — *purpureum*, iii. 139  
 — *violaceum*, iii. 139, 140  
*Trichophytosis*, iii. 138  
*Trichuris trichiura*, ii. 126  
 Tricuspid lesions, i. 434  
 Trinitrotoluene, poisoning by, ii. 602  
 Trional, poisoning by, ii. 602  
 Trophœdema, ii. 401  
 Troussau's sign, iii. 341  
*Trypanosoma castellanii*, iii. 366, 367  
 — *gambiense*, iii. 366, 367  
 — *rhodesiense*, iii. 367  
 — *vivax*, iii. 367  
*Trypanosomiasis*, African, iii. 366  
 — South American, iii. 371  
 Tuberculin, in diagnosis, iii. 190  
 — in treatment, iii. 50  
 Tuberculosis care committees, iii. 393  
 — clinical classification of, iii. 382  
 — cutis orificialis, iii. 206  
 — declining death-rate from, iii. 379  
 — dispensary, iii. 387  
 — environmental influence in, iii. 375  
 — experimental inoculation of, iii. 374  
 — historical notes on, iii. 374  
 — in households, iii. 382  
 — incidence of, iii. 380  
 — local effects of, iii. 375  
 — mortality statistics of, iii. 376  
 — notification and, iii. 387  
 — prevalence of, iii. 376  
 — prevention of, iii. 383  
 — schemes, iii. 386  
 — suitable employments for subjects of, iii. 394  
 — susceptibility to, iii. 383  
 — systemic effects of, iii. 376  
 — verrucosa, iii. 206  
 Tuberculous adenitis of suprarenals, iii. 330  
 Tuho-ovarian cysts, ii. 445  
 Tubular diarrhoea, i. 260  
 Tuning-fork tests, i. 401  
 Turbinal bones, enlargement of, ii. 335  
 Turkish bath, ii. 53  
 Tylosis, ii. 333  
 Typhoid spine, iii. 405  
 Typhoidin, iii. 192  
 Typho-protein, iii. 192  
 Tyrosin in urine, iii. 455

UHLENHUTH's test, i. 176  
 Ulcerative colitis, i. 263  
 — dermatitis, iii. 510  
 — tuberculous peritonitis, ii. 528  
 Ulcers from extensive burns, iii. 420

Ulcers, trophic, iii. 420  
 Ulcus serpens, i. 293  
 Ulnar-nerve palsy, iii. 247  
 Umbilical hernia, ii. 33  
*Uncinaria americana*, ii. 126  
 — *duodenalis*, ii. 126  
 Unilobular cirrhotis, ii. 215  
 Urates in urine, iii. 454  
 Urea concentration test, ii. 340  
 — in urine, iii. 449  
 Ureters, catheterization of, i. 313  
 Urethra, foreign bodies in, ii. 493  
 Urethral dilatation, iii. 430  
 Uric-acid crystals in urine, iii. 454  
 Urinary cysts, examination of contents of, i. 155  
 — filaments, iii. 455  
 Urine, aceto-acetic acid in, iii. 444  
 — acetone bodies in, iii. 444  
 — acid calcium (stellar) phosphate crystals in, iii. 454  
 — — — sodium urate in, iii. 454  
 — albumin in, iii. 451  
 — albumoses in, iii. 452  
 — amino-acids in, iii. 449  
 — ammonia in, iii. 449  
 — ammonium urate in, iii. 451  
 — ammonium - magnesium ("triple") phosphate crystals in, iii. 454  
 — "bacterial casts" in, iii. 455  
 — bases in, alloxuric, iii. 450  
 — — — nuclein, iii. 450  
 — — — xanthin, iii. 450  
 — Bence-Jones albumose in, iii. 452  
 — — — protein in, iii. 453  
 — beta-oxylbutyric acid in, iii. 445  
 — bile acids in, iii. 454  
 — — — pigments in, iii. 453  
 — calcium carbonate in, iii. 455  
 — — — oxalate crystals, iii. 454  
 — — — sulphate in, iii. 455  
 — casts in, iii. 455  
 — chlorides in, iii. 442  
 — cholesterin in, iii. 455  
 — — — chromogen in, iii. 452  
 — colour of, iii. 441  
 — creatin in, iii. 450  
 — creatinin in, iii. 450, 455  
 — cylindroids in, iii. 455  
 — cystin in, iii. 455  
 — dextrins in, iii. 448  
 — dextrose in, iii. 445  
 — diacetic acid in, iii. 444  
 — earthy phosphates in, iii. 454  
 — epithelial cells in, iii. 455  
 — erythrocytes in, iii. 455  
 — estimation of sugar in, iii. 447  
 — fat in, iii. 456

Urine, fatty-acid crystals in, iii. 456  
 — ferments in, iii. 454  
 — filaments in, iii. 455  
 — fructose in, iii. 446  
 — glucose in, iii. 445  
 — glucuronic acid in, iii. 447  
 — hæmatoidin in, iii. 455  
 — hæmatoporphyrin in, iii. 452  
 — hippuric acid in, iii. 455  
 — homogentistic acid, iii. 447  
 — — — hydrobilirubin in, iii. 452  
 — — — indican in, iii. 441  
 — — — iso-glucuronic acid in, iii. 446  
 — — — lactose in, iii. 446  
 — — — lævulose in, iii. 446  
 — — — leucin in, iii. 455  
 — — — leucocytes in, iii. 455  
 — — — magnesium phosphates in, iii. 455  
 — — — mucus in, iii. 455  
 — — — nitrogen output in, total, iii. 448  
 — — — oxalic acid in, iii. 444  
 — — — pancreatic reaction in, iii. 448  
 — — — pentoses in, iii. 447  
 — — — phosphates in, iii. 442  
 — — — pseudo-lævulose in, iii. 446  
 — — — pus in, iii. 455  
 — — — reaction of, iii. 442  
 — — — skatol in, iii. 444  
 — — — specific gravity of, iii. 411  
 — — — sulphates in, iii. 413  
 — — — tyrosin in, iii. 455  
 — — — urates of sodium and potassium in, iii. 454  
 — — — urea in, iii. 449  
 — — — uric acid in, iii. 450, 451  
 — — — urobilin in, iii. 452  
 — — — urobilinogen in, iii. 452  
 — — — urochrome in, iii. 452  
 Urobilinogen in urine, iii. 452  
 Urochrome in urine, iii. 452  
 Uterine adenomyomata, iii. 471  
 — carcinoma, cervical, iii. 473  
 — — — corporeal, iii. 472  
 — — — fibroids, calcification of, iii. 464  
 — — — cervical, iii. 462  
 — — — complicating pregnancy and labour, iii. 465  
 — — — corporeal, iii. 461  
 — — — degenerations of, iii. 464  
 — — — interstitial, iii. 462  
 — — — ligamental, iii. 462  
 — — — submucous, iii. 462  
 — — — subperitoneal, iii. 461  
 — — — treatment of, iii. 470  
 — — — polypi, fibroid, iii. 476  
 — — — malignant, iii. 478  
 — — — mucous, iii. 477  
 — — — placental, iii. 477  
 — — — sarcoma, cervical, iii. 475  
 — — — corporeal, iii. 471  
 — — — souffle, iii. 3

## INDEX OF DETAILS

- VACCINATION**, prophylactic, ii. 73  
 ——— therapeutic, ii. 74  
**Vaccine**, cholera, ii. 74  
 ——— dysentery, ii. 74  
 ——— plague, ii. 74  
 ——— pneumococcus, ii. 74  
 ——— typhoid and paratyphoid, ii. 73  
**Vaccines** as colloids, i. 269  
**Vagina**, inversion of, from above downwards, ii. 490, 500, 501  
 ——— laceration of, ii. 160  
**Vaginal hydrocele**, ii. 44  
**Vapour bath**, ii. 53  
 "Varicose eczema," i. 411  
**Vegetable irritants**, i. 407  
**Venous sinuses**, injuries of, ii. 4  
**Ventral hernia**, ii. 33  
 — — horns, localization of lesions of, iii. 234  
**Vernon Harcourt's inhaler**, i. 69  
**Vesicular moles**, ii. 624  
**Vestibular reaction tests**, i. 403  
**Vibrio cholerae**, i. 146, 242  
**Vichy waters**, iii. 225  
**Victoria Dispensary**, Edinburgh, iii. 388  
**Vipera ammodytes**, iii. 221  
 ——— *aspis*, iii. 221  
 ——— *berus*, iii. 221  
 ——— *russelli*, iii. 221  
**Viperines**, iii. 221  
**Vital's test for pus in urine**, iii. 455  
**Vittel waters**, iii. 226  
**Von Noorden's diet**, ii. 397  
**Vulvitis**, iii. 524  
**WALLERIAN degeneration**, ii. 365  
 "Washerwoman's eczema," i. 408, 413  
**Waters** (*see* Mineral waters)  
**Water-soluble B**, iii. 516  
 ——— C, iii. 518  
**Waxy degeneration**, i. 52  
**Weber's test**, i. 402  
**West's swab**, i. 147  
**Wet-nursing**, ii. 96  
**Wheal of urticaria**, iii. 158  
**Wiesbaden waters**, iii. 221  
**Wildungen waters**, iii. 226  
**Wills**, capacity for making, iii. 336  
 "Window screens," iii. 74  
**Wohlfartia magnifica**, ii. 319  
**Wohlgemuth's method of estimating ferments**, iii. 454  
**Wolfian cysts**, iii. 487  
**Woodhall Spa waters**, iii. 224  
**Working colonies**, iii. 393  
**Woulfe's bottles**, ii. 559  
**Wright treatment of wounds**, iii. 539  
**Wrist - joint, sprains of**, iii. 253  
**XANTHIN** bases in urine, iii. 450  
**Xanthochromia**, iii. 239  
**YERSIN-ROUX plague serum**, ii. 551  
**ZIEHL-NEELSEN's method**, i. 146  
**Zinc acetate test for urobilinogen in urine**, iii. 452  
**Zone phenomena**, ii. 68  
**Zymotic diarrhoea**, i. 358

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